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In Memoriam

HERMON BROOKFIELD VAN WYCK

B.A., M.B., F.R.C.S.(C), F.R.C.O.G.

(1890-1952)

. . . . Dr. Van Wyck was born April 24, 1890, in St. Catharines, Ontario, in the Methodist parsonage, the second of two sons of the Rev. and Mrs. James Van Wyck.

In 1907 he matriculated with honors from Harbord Collegiate, being awarded the first Edward Blake Scholarship in languages. He then enrolled in Honor Classics in Victoria College from which he obtained in 1911 his B.A., University of Toronto. He entered the Medical School of the University of Toronto in 1911 and was granted his M.B., 1915, while serving as a non-commissioned officer with No. 4 University of Toronto, Canadian General Hospital. With this unit he served throughout the First War in Salonika and England, returning with it to Canada as Major and Adjutant for demobilization in September 1919.

Following discharge from the army, he began post graduate training in Obstetrics and Gynecology in the Toronto General Hospital under Dr. B. P. Watson. He was then associated with the late Professor V. J. Harding for three years as a fellow in Obstetrics and Gynecology in research on the toxaemias of pregnancy. They were among the first of those to point out the role of salt restriction in the treatment of pre-eclamptic toxaemia. He then entered private practice in obstetrics and gynecology and became a junior staff member in this department at the Toronto General Hospital and this University. In 1946 he was appointed Chief of the Obstetrical and Gynecological service in the Toronto General Hospital, and Professor and Head of the Department of Obstetrics and Gynecology in this University. On his retirement in 1950, he was appointed Professor Emeritus in Obstetrics and Gynecology and at the same time retained his active role in the Faculty as Special Lecturer in the Humanities in Medicine.

Besides carrying on a large and extensive private practice, he was an excellent teacher and lecturer and was always anxious and eager to lend a helping hand to all or any he might think were in need. He particularly loved contact with students and the younger men who found in him great inspiration and encouragement. . . . .

Professor D. M. Low in a resolution to the Council of the Faculty of Medicine of the University of Toronto, April 4, 1952.

Dr. Van Wyck was for many years closely associated with this Medical Journal and took a deep interest in all its activities. In his death, the Journal and the student body as a whole have lost a regular contributor, a trusted guide, and a true friend.

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SOME NEUROLOGICAL ASPECTS OF BLADDER FUNCTION

W. G. GRANT, M.D.*

Disturbance of bladder activity is often an early and serious manifestation of disease of the nervous system. If recognized and treated early much may be done to prevent permanent disability and to avoid the complications which increase morbidity in the chronically ill patient.

The following discussion is based upon information from the literature and from the experience of clinicians in this centre. There has been a fuller understanding of the subject in recent years, much of which was obtained from experience in the care of paraplegics from the last two wars. In World War I only 10% of American soldiers with spinal cord injuries survived over a year; over 90% of those from World War II were still living in 1948 (2). The urological complications of spinal cord injury and disease were the most important causes of death in both groups.

NORMAL ANATOMY AND PHYSIOLOGY

Bladder Musculature

The muscle fibres of the bladder wall make up the detrusor muscle. Over the fundus of the bladder these fibres are arranged in an irregular interlacing pattern. This makes it possible for the bladder to contract completely and obliterate its cavity. Below the level of the ureteral orifices the bulk of the fibres run longitudinally downward in the wall of the bladder neck and prostatic urethra, and end at the level of the lower end of the prostate. When these contract they pull the prostatic urethra up and shorten it, and at the same time they widen the bladder neck and the urethral orifice of the bladder (8).

Around the membranous urethra lies the external sphincter, formed by the muscle of the urogenital diaphragm. This sphincter is under voluntary control, but relaxes reflexly when urine enters the prostatic urethra (1, 8).

Nerve Supply

Although the bladder has both parasympathetic and sympathetic connections, the essential innervation is through the parasympathetic (6, 7, 8).

a) *Parasympathetic*—The parasympathetic supply is through the pelvic nerves, which arise from the second, third, and fourth sacral segments. The

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pelvic nerves contain motor fibres to the detrusor and afferent fibres giving rise to sensations of fullness, pain, temperature, and touch. These fibres form the reflex arc governing micturition, and they synapse in the cord to make connections with the cortex.

b) *Sympathetic*—The presacral nerve, which is formed from the hypogastric plexus, carries sympathetic fibres to the bladder. These are both preganglionic and postganglionic, the former synapsing in the wall of the bladder, and the latter coming from the hypogastric ganglia. Their spinal origin is in the 11th and 12th dorsal and 1st lumbar segments (7). Sympathetic nerves play no essential part in micturition. Their main function in humans is in regulating the blood supply to the bladder, and in the transmission of certain vague visceral sensations (1, 7, 8).

c) *Somatic*—The internal pudendal nerves (S-2, 3, 4) supply voluntary motor fibres to the external sphincter and the superficial muscles of the perineum. They carry cutaneous sensation from the saddle area of the perineum, most of the external genitalia, and the mucosa of the urethra (6).

d) *Cortical*—Bladder sensation is directed to the sensory cortex through the posterior columns and lateral spinothalamic tracts. The frontal lobes of the cortex contain an inhibitory centre, which has connections with the sensory and motor cortex. The motor cortex is connected through the pyramidal tracts with the somatic and parasympathetic motor cells in the sacral cord.

The cortex has two influences over micturition. First and most important is the inhibitory influence of the frontal and motor cortex over the spinal reflex. This inhibition is below the level of consciousness until the bladder has reached near capacity. The second is the voluntary control over the external sphincter (6, 7). This muscle is used to prevent voiding when there is painful distension, and is the only means of control in certain types of neurogenic bladder (see below).

The Act of Micturition

Micturition in the normal adult may be considered as a spinal reflex which is under the influence of higher centres.

The kidneys secrete from 1-2 cc of urine per minute into the bladder. When bladder content reaches about 150 cc a feeling of fullness is appreciated (3, 8). This sensation passes via the pelvic nerves to the sacral cord, and thence to the sensory cortex and the inhibitory centre in the frontal lobes. The inhibitory influence of the cortex, acting on the sacral segments of the cord, allows the bladder to be filled to between 400 and 500 cc (6, 7, 8). At this stage the sensation of fullness becomes painful; the inhibitory effect is no longer able to depress the lower centres, and uncontrolled bladder contraction takes place. Voiding may be voluntarily started at any point in the filling of the bladder.

At the start of micturition the detrusor contracts. This shortens and widens the bladder outlet and sharply raises the intravesical pressure. Contraction of the pubo-coccygeus fibres, running downward and backward from the prostate, tend to draw the prostate down and back, thus further widening the urethra. Urine is forced into the prostatic urethra. Relaxation of the external sphincter is brought about by a spinal reflex initiated by the flow of urine through the

urethra. This reflex is mediated by the internal pudendal nerves, which also cause the bulbocavernosus to contract at the end of micturition (1). Contraction of the abdominal muscles is not necessary for normal bladder evacuation (3, 4, 6).

Attributes of the Normal Bladder

1. Full voluntary control of micturition.
2. Full bladder sensation.
3. Capacity 400-500 cc.
4. Absence of uninhibited contractions during filling (see below).
5. No residual urine.
6. Intravesical pressure from 1-25 cm. water. Average 8-15 cm. Normal bladder can accommodate for different volumes with very little change in pressure.

Cystometry

Detailed accounts of the procedure of cystometry can be found in most recent textbooks of urology. The general principles are outlined here for those not familiar with them. The cystometrogram is essentially a graph of the intravesical pressure plotted against volume of fluid in the bladder. Bladder capacity, bladder contractions, sensation, and degree of voluntary control can be accurately assessed.

An indwelling catheter is inserted, and this is connected as shown (Fig. I) with a reservoir of sterile fluid and an inverted T-tube, in a continuous system. The base of the inverted T-tube is on a horizontal level with the bladder. A capillary tube is attached to the free end of the T-tube. The bladder is drained, and the system is emptied of air by running fluid through it. The overflow outlet is then clamped. It will be seen that now, as pressure in the bladder rises the level of fluid will rise in the T-tube, and the pressure may be read as a direct measurement in cm. of fluid.

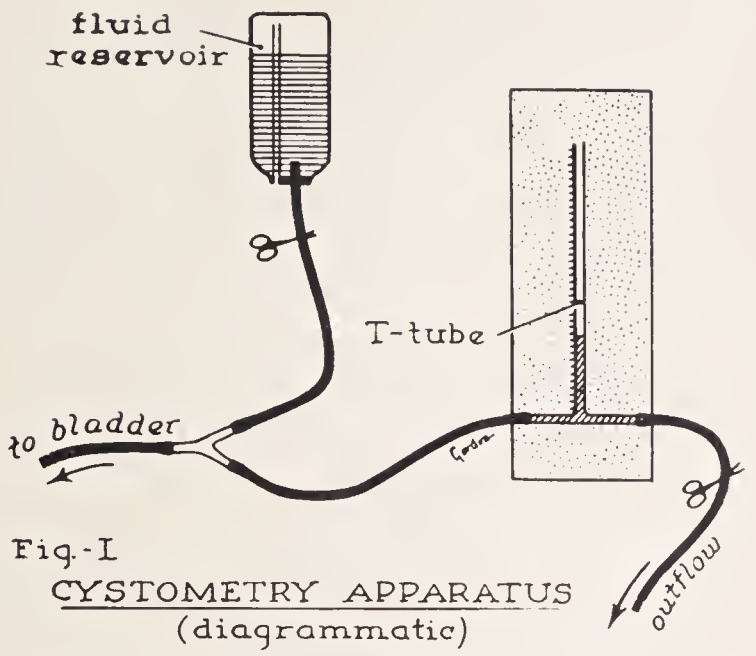

Fig.-I
CYSTOMETRY APPARATUS
(diagrammatic)

With the bladder empty the height of the column of water, which will be near zero, is taken as the starting point. Now fluid is run into the bladder slowly from the reservoir, 50 cc at a time. When the fluid level in the T-tube is resting at a constant level after each 50 cc increment, a pressure reading is made and recorded. Any involuntary contractions are recorded in terms of maximum pressure attained.

When bladder capacity is reached, there is an uncontrolled contraction which sends the fluid column over the top of the T-tube. This causes some discomfort, and the outlet is at this point opened and the bladder allowed to drain. More than 500-600 cc should not be used in filling the defective bladder because of danger of damage to the muscle.

The information is charted on a graph. The following points are of value, and should be indicated on the cystometrogram:

1. Involuntary bladder contractions.
2. Point at which discomfort occurs, or other phenomena such as sweating or flushing.
3. Medication—atropine, diuretics, sedatives, etc.
4. State of rectum—full or empty.
5. Time—repeated cystometrograms should be done at about the same time each day, because of differences noted before and after meals, etc.

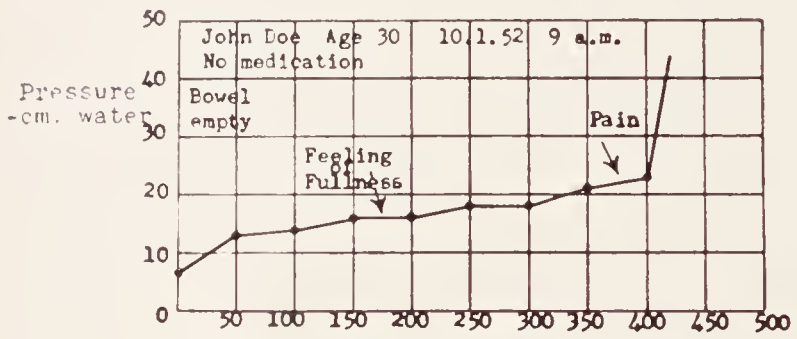

Fig.II - Cystometrogram. Normal bladder.

THE NEUROGENIC BLADDER

When the function of the bladder is altered because of disease of the nervous system, it may be termed a neurogenic bladder. Where the lesion disrupts the spinal reflex arc a so-called "lower motor neurone bladder" results. Damage above this level produces an "upper motor neurone bladder". Since the spinal reflex is mediated through segments S-2, 3, 4, it would be supposed that any cord lesion above this level would cause an upper motor neurone bladder. However, traumatic lesions to the cord higher up (e.g.—fracture as high as the 12th thoracic vertebra) *may* result in a lower motor neurone type of bladder. The reason for this is not known. In slowly developing cord or nerve disease the resulting bladder dysfunction corresponds to the anatomical site of the lesion.

Considering again that normal micturition is brought about by sacral spinal reflex activity which is under cortical control, the results of complete cord transection at higher levels are understandable. Micturition is then carried on entirely as a spinal reflex. Lesions lower down, on the other hand, will disrupt the spinal reflex, but the exact type of bladder dysfunction will depend upon what part of the reflex arc is damaged.

Types of Neurogenic Bladder

There is no universal classification of the neurogenic bladder. Clinicians dealing with organic neurology see affections due to syphilis, poliomyelitis, multiple sclerosis, etc. Those caring for paraplegics encounter the results of acute traumatic lesions. The following scheme is an attempt to deal with all these types.

A. Upper Motor Neurone Bladder
   1. Uninhibited bladder
   2. Reflex bladder

B. Lower Motor Neurone Bladder
   1. Autonomous bladder
   2. Sensory paralytic bladder
   3. Motor paralytic bladder

C. Bladder Following Acute Cord Trauma

A. *Upper Motor Neurone Bladder*

1. Uninhibited Bladder

This is seen in lesions of the frontal lobe or motor cortex, such as localized tumour or cerebrovascular accident. A congenital type is occasionally seen in otherwise normal people.

The function lost is that of cortical inhibition of the reflex. All other faculties are retained, including voluntary control of the external sphincter. As the bladder fills it undergoes mild contractions under the stimulus of distension. These increase until a massive contraction occurs, emptying the bladder. Sub-

jectively, each of these minor contractions is interpreted as a need to void, but the voiding contraction does not take place until around 100-150 cc are in the bladder. To a great extent, incontenence is avoided by the forcible voluntary contraction of the external sphincter. The result is urgency and precipitancy, more or less well controlled.

Essentials of this type of bladder are:

1. Full bladder sensation.
2. Loss of cortical inhibition of the spinal reflex.
3. Voluntary control over the external sphincter.
4. Uninhibited bladder contractions during filling.
5. Reduced capacity.
6. No residual urine.

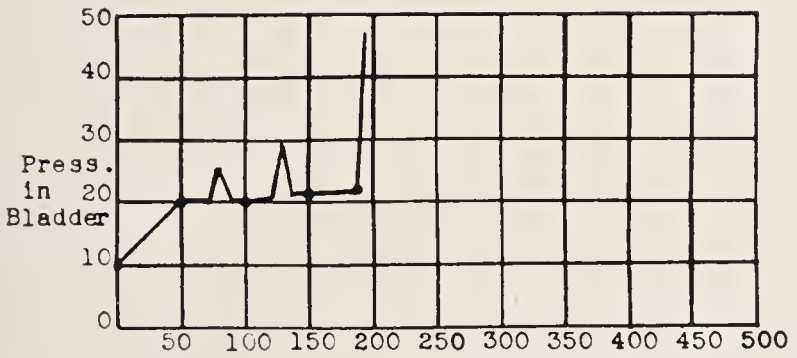

Fig. III - Cystometrogram. Uninhibited Bladder

2. Reflex Bladder

This type results from a complete lesion of the upper motor neurone. It is also the type of bladder possessed by the normal infant. In the latter the supra-segmental connections are not fully developed; in the former they have been destroyed. The result is a bladder which functions independently, by means of a spinal reflex. There is no cortical control.

The cause may be a more extensive lesion of the type seen in the uninhibited bladder, or anything causing transection of the cord above the level of the second sacral segment.

Characteristics are:

1. Complete loss of normal bladder sensation.
2. Loss of all voluntary control over micturition.
3. Uninhibited contractions during filling.
4. Reduced bladder capacity. If repeated infection is avoided and proper tidal irrigation used, a small, scarred, contracted bladder does not develop.
5. Residual urine of 25-100 cc.

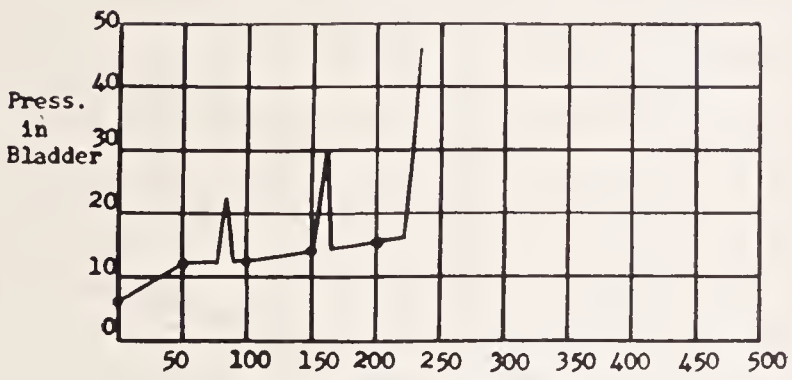

Fig. IV - Cystometrogram. Reflex Bladder

B. *Lower Motor Neurone Bladder*

The syndrome produced by lesions of the lower motor neurone depends upon whether the cord itself, or the sensory or motor limbs of the reflex arc are affected. The following emerge as definite clinical pictures.

1. The Autonomous Bladder

This is seen when the function of the whole reflex is destroyed by complete lesions of the sacral cord or pelvic nerves. Trauma, neoplasm, inflammation, etc., may be the primary cause. It is seen in normal subjects during spinal anaesthesia.

This bladder, if overdistension is avoided, will develop a certain tone, al-

though completely separated from extrinsic nervous connections. The cystometrogram may show weak contractions during filling, but these are not sufficient to cause emptying. This tone is believed to be due to ganglionic plexuses within the bladder wall. The bladder can be likened to a rubber balloon, the tension within it increasing steadily as it fills.

The capacity is difficult to determine, since there is neither voluntary nor reflex evacuation, and sensation is lost. Voiding is by overflow incontinence, unless this is avoided by periodic pressure on the abdominal wall.

Characteristics:

1. Loss of normal bladder sensation.
2. Absence of both voluntary and reflex voiding.
3. Residual urine 50-250 cc.

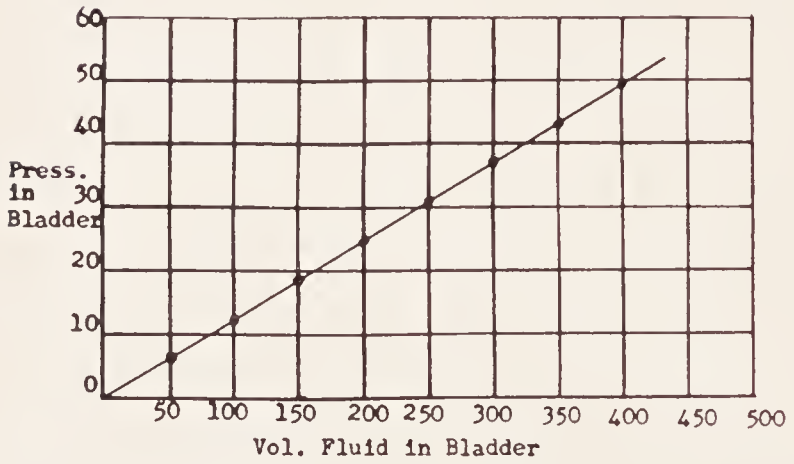

Fig.V - Cystometrogram. Autonomous Bladder

2. Sensory Paralytic Bladder

The classic example of this is seen in the tabetic. Any lesion destroying the posterior nerve root function and sparing the motor root will lead to sensory bladder paralysis. The bladder is nearly always large, flabby, and atonic. The atony is partly due to frequent and prolonged overdistension, the patient being unaware that the bladder is full. Similar lesions produced experimentally will not result in atony if catheter drainage is started early (6). If the disease later advances to involve the motor root as well, an autonomous bladder may result, indicating that some factor besides overdistension is responsible for the atony.

No adequate explanation for the two types of bladders resulting from interruption of the reflex arc, one on the sensory side and one on both motor and sensory, has been observed (6).

Voiding is by overflow incontinence unless the patient is trained to expel some urine at intervals by manual pressure over the abdomen. No reflex voiding can be initiated, and the pressure must be maintained to keep the urine flowing.

Characteristics:

1. Complete absence of normal bladder sensation.
2. Loss of voluntary and reflex micturition.
3. Voiding by overflow or abdominal pressure.
4. Very large capacity—sometimes 800 cc or more.
5. Large residual urine.
6. Low intravesical pressure.

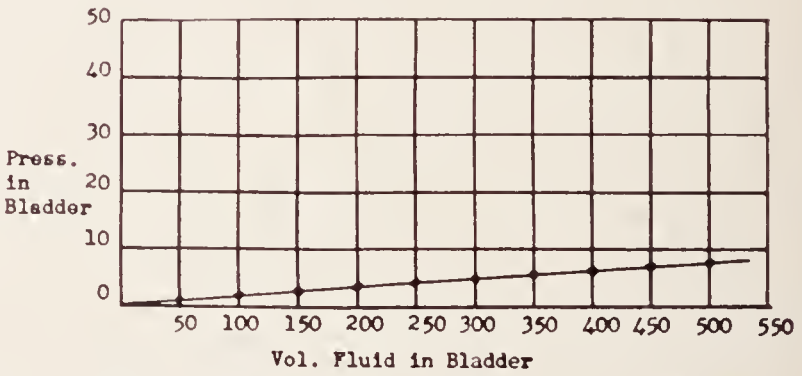

Fig.VI - Cystometrogram. Sensory Paralytic Bladder

3. Motor Paralytic Bladder

Anterior poliomyelitis, neuromyelitis, and other conditions in which the efferent nerve alone may be injured, will cause this type of dysfunction. It is also seen during recovery from a general anaesthetic.

Bladder sensation is intact, and the patient is distressingly aware of a full bladder, but cannot void. Neither voluntary nor reflex micturition is possible.

In nearly all cases complete recovery follows, the disability lasting from days to months. Permanent damage and an atonic bladder may result from overdistension.

Characteristics:

1. Normal sensation.
2. Absence of both voluntary and reflex micturition.
3. Approximately normal capacity.

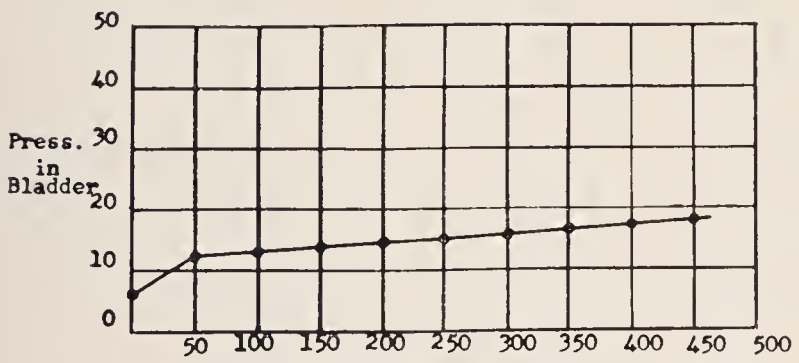

Fig.VII - Cystometrogram. Motor Paralytic Bladder

C. *Bladder Following Acute Cord Trauma*

In spinal cord injury at any level a state of spinal shock immediately occurs, with atony and absent limb reflexes below the level of the lesion. This condition may last from a few hours to several months, and if the patient survives, there is eventual progression to a syndrome in keeping with the level and extent of the cord damage. The state of the bladder roughly corresponds to the state of the lower limb muscles.

During the early phase the bladder is atonic and insensible. It distends as a flaccid bag, and may be damaged if not drained early. As bladder function returns, small myotonic contractions are seen on the cystometrogram. At about the stage where the plantar response begins to reappear, the tone of the bladder usually returns. Thus, when daily stroking of the sole of the foot first reveals an upgoing toe, the clinician knows that tidal irrigation should be started. To persist with straight catheter drainage beyond this time is to invite a low-capacity bladder.

Whether complete recovery ensues or some type of neurogenic bladder persists, the lower motor neurone function is the first to return, in the form of an autonomous bladder. If the lesion is high in the cord, a reflex bladder will later develop.

SUMMARY

The functional anatomy and the physiology of the bladder are reviewed in the light of recent findings. A brief description of bladder innervation is given and correlated with the various types of bladder dysfunction seen clinically in lesions of the nervous system.

ACKNOWLEDGMENTS

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ELECTROMYOGRAPHY

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In the past decade the technique of electromyography has established its importance and usefulness. The reason for its having been relatively neglected during the years when electrocardiography and electroencephalography were proving more and more indispensable is simply this: no reliable apparatus was available. With the rapid advance of electronics accompanied by the urgent needs of war surgery, new methods were quickly developed for accurate measurement of the small electrical changes in muscle.

Although the new interest in electromyography was at first confined to neurologists and physical therapists, soon this new method was being reinvestigated by physiologists and anatomists. Unfortunately, enthusiasm for a new method produced some reports which were premature and others which were erroneous. This does not detract from its present usefulness.

Electromyography is the science of measuring the electrical changes that take place in a muscle. These changes, known as "action potentials", are due to the activity of muscle cells or fibres and they may be picked up by various types of electrodes which have different qualities. For example, skin electrodes of the type used in electroencephalography have been used widely. These electrodes are very convenient, painless, and give a general picture of the electrical changes that are occurring in surface muscles. However, skin electrodes are not sufficiently reliable for accurate work.

Various types of needle electrodes are being used. Common examples are: two needles situated various distances apart; a needle electrode paired with a near-by skin electrode; and the "concentric needle" electrode. The last consists of an hypodermic needle with an insulated metal core—usually a fine wire. The barrel of the needle acts as one electrode; the core acts as the other. As a rule, all types of needle electrodes are insulated except at their very tips. The advantage of needle electrodes lies in their much greater accuracy. An obvious disadvantage is the discomfort to the subject. This is actually not as great as one would think and our subjects, with few exceptions, have rarely complained. Another, less obvious, objection to needle electrodes is that for some investigations they are *too* localizing and accurate.

The investigator, then, has available many kinds of electrodes which he can adapt to his own particular problem.

Since the action potentials are so small—they measure in millivolts and microvolts—a reliable amplifier is essential. These are now available commercially. Some are especially designed for electromyography; others are modified electroencephalographs. Many investigators simply use an unaltered elec-

troencephalograph. Of these there are various types which are of varying accuracy.

To record the electrical changes there are, again, various methods. Those who use e.e.g. equipment usually record on e.e.g. paper with ink-writing pens. Since this is a mechanical system, great care must be exercised to avoid artefacts in recording. A considerable body of literature has appeared about these problems alone.

The cathode ray oscillograph is ideal for visualizing action potentials because the electron stream has very little inertia and distortion is minimal. With the cathode ray, various photographic methods are used to make permanent records on film or paper.

Most investigators also incorporate in their equipment a loud speaker which translates the electrical changes into sound. Some workers have elaborated methods of recording this sound on magnetic tape or wire so that it may be reproduced at a later date. Several centres are busy building more and more complex and wonderful electronic gadgets along these lines.

Although electromyography may be said to date back to the first experiments of Galvani and Volta in the 18th century, the significant history of modern electromyography begins with the work of Adrian and Bronk at Oxford. These physiologists developed the concentric needle electrode and published their classic work on electrophysiology in 1929.

The physiologist already knew that normal muscle was made up of motor units. A motor unit is a group of muscle cells or fibres—from several to several hundred—all supplied by one motor nerve fibre. When an impulse travels down the nerve fibre it causes all of the muscle fibres in its motor unit to contract

It was soon apparent that the needle electrodes of Adrian and Bronk were picking up the electrical changes in individual motor units as they contracted. Such changes ranged from 100 microvolts to one or two millivolts. (A microvolt is a millionth, a millivolt is a thousandth of a volt).

In 1944, Weddell, Feinstein and Pattle at Oxford published the results of their study on human beings with a variety of neuromuscular dysfunctions, especially traumatic nerve lesions. They showed that all limb muscles can be completely relaxed so that there is no electrical activity. Their description of normal and abnormal wave forms has formed the basis of all subsequent investigation.

At the Montreal Neurological Institute, Captain Herbert H. Jasper, R.C.A.M.C., and his colleagues rapidly developed equipment and methods for evaluating nerve lesions in army casualties. The Jasper group introduced the unipolar needle which, in some clinics, has replaced all other electrodes.

The Scandinavian investigators were soon publishing many important results, and the names of Buchthal, Kugelberg, and Clemmesen figure prominently in the literature. Bauwens and Richardson, at St. Thomas's Hospital, London, are adding new knowledge to clinical electromyography.

In the realm of anatomical investigation the first real contribution was made by Inman, Saunders and Abbott of California, who worked out many of the problems of muscle function for the shoulder region. Their paper has been

followed by others on the muscles of the arm, wrist, back, abdomen and hip. Dr. R. E. Moyers, now Professor of Orthodontics in the University of Toronto, was the first to apply electromyography to the study of the muscles of mastication.

Electromyography, then, falls into two general classes: clinical and experimental. Clinical electromyography, being still a new science, contains a high proportion of experiment. Accordingly, there is some overlapping of the two classes.

Clinical Electromyography

Investigation

Physical therapists and neurologists are investigating the changes that occur with various motor nerve lesions. Poliomyelitis has received the chief emphasis for obvious reasons. Primary and secondary muscle dysfunctions are being thoroughly studied in many clinics, as are the compressions of the spinal cord and nerve roots.

Diagnosis

Using the new knowledge gained by clinical investigation many medical centres are calling upon electromyography to aid in diagnosis. Indeed, in several states of the U.S.A. electromyographic results have been accepted as evidence in court of law. In Toronto, the Hospital for Sick Children has an electromyograph primarily for research, but it has been used on occasion for diagnosis. The Department of Electroencephalography under Dr. John Scott at the Toronto General Hospital, is prepared to aid in clinical diagnosis. At the Malton Convalescent Hospital, Dr. Zinovieff is in charge of a new electromyograph obtained by the Workmen's Compensation Board. Sunnybrook Veterans' Hospital also has electromyographic equipment.

Clinical Findings

A normal muscle can be put at complete rest resulting in the complete absence of electrical activity. The screen of the cathode ray tube shows a straight base line and the loud speaker is quiet. When the muscle contracts, either voluntarily, reflexly or on artificial stimulation of its nerve, the base line is disturbed by a series of waves—usually diphasic—and there is a loud crackling noise from the loud speaker. These persist until the muscle is completely relaxed again.

If the muscle has been completely denervated by either trauma or by disease of the lower motor neurone, the early picture is one of complete "silence". After eighteen days there appear some very fine contractions in the muscle which are both invisible and impalpable on clinical examination. These are known as fibrillations and they appear as tiny deflections of the base line. Whereas the normal motor unit contraction ranges from 100 to 2000 microvolts and has a duration of 5 to 10 milliseconds, a fibrillation potential is well below 100 microvolts in intensity and around 1 or 2 milliseconds in duration. The loud speaker gives a fine crackling sound which is easily recognized by the experienced observer.

Fibrillation should not be confused with fasciculation, the term applied by Denny-Brown to the easily observable fine quivering of muscle. Many normal individuals have observed fasciculation in their own muscles, especially the thenar group, and have been unnecessarily concerned. It must be noted, how-

ever, that fasciculation is of some diagnostic significance during recovery of nerves after interruption. Its e.m.g. picture is one of scattered motor unit contractions.

Considerable importance is attached to the presence or absence of fibrillation in a case of denervation, both from the standpoint of diagnosis and of prognosis. There is some disagreement on the significance of fibrillation in prognosis and no purpose would be served in a paper of this nature to enter the controversy. Warming a muscle or the injection of prostigmine elicits a greater number of fibrillations in a denervated muscle.

Other abnormal wave forms are encountered notably during the recovery phase of a nerve lesion and in poliomyelitis. These have been well-described and their significance has been well-established. Some clinics emphasize the occurrence of bursts of potentials on insertion of the needle—"insertion-potentials"; others emphasize a phenomenon known as "synchronization". This latter, which appears in lower motor neurone lesions, is found where two or more separate leads are taken from the same muscle. On minimal voluntary contraction the action potentials from these leads are synchronous. Denny-Brown has suggested that the multiple leads are simply registering the potentials from a single motor unit that has been laid bare by the silence of the surrounding motor units.

Acute Anterior Poliomyelitis: A completely paralysed muscle in poliomyelitis is at first silent, but after about three weeks it shows fibrillation potentials. These fibrillations may persist for many years after the acute disease. Partially paralysed muscles show a mixed picture of the normal and the abnormal. Prognosis should be especially significant in electromyography of this disease and standards are being set up by which it is hoped that the outlook for recovery during the convalescent stage can be determined. The early recovery of motor units often can be detected before there is any clinical evidence of power in a muscle.

Other Diseases of the Spinal Cord: Degenerative diseases such as early amyotrophic lateral sclerosis, multiple sclerosis, subacute combined degeneration and progressive muscular atrophy all show signs electromyographically. Various other conditions are being investigated such as tabes dorsalis, herpes zoster and bacterial infections.

Pressure on the Spinal Cord and Nerve Roots: Cord tumors and extruded intervertebral discs have been localized accurately by electromyography. Some centres have perfected techniques whereby the diagnosis of "a disc" can be confirmed before operation. The usefulness of this procedure has been especially well demonstrated in the cervical region.

Peripheral Nerve Lesions: If, on clinical examination, the muscles supplied by an injured nerve show no activity, but they do show some electromyographic activity, the prognosis for recovery is good, for, obviously, the continuity of the nerve is still maintained. Complete electromyographic silence followed by fibrillations in three weeks suggests a complete lesion and the surgeon does not delay suturing the nerve. The progress of recovery with or without an operation can then be followed by the electromyograph.

Primary Muscle Diseases: "Insertion potentials" are characteristic of the myo-

tonias (congenita, dystrophica, and acquisita). No fibrillations are seen. Voluntary contractions produce normal waves which persist for some time instead of disappearing immediately on relaxation.

Myasthenia gravis mirrors the changes seen clinically when the patient performs a series of movements. That is, the electrical activity, normal at first, diminishes progressively. Clinical and myographic recovery occur together.

Tremor: Some interesting work has been done by Clare and Bishop with Price and Mills, and by Hoefer and Putnam on the subject of tremors. Some efforts have been made by other investigators to determine whether a tremor of organic origin can be differentiated from one that has been artificially induced. At present, more work is needed before electromyographic evidence would be fully acceptable in this field.

Basic Scientific Research

PHYSIOLOGY

The physiologists have added modern electromyography to the tools with which they are studying muscle function. Their chief interest has been in learning more of the responses of the motor unit and their work has overlapped that of the clinicians. Many experiments on animals are being conducted with the practical problems of nerve-muscle physiology in mind. In spite of this, it must be admitted that the initiative in electromyography seems to have been forfeited by the physiologists.

ANATOMY

Heretofore, what we have known about muscle function has been based on physical examination of the muscles in action, observation of the changes in paralyses, application of general mechanical principles, and the exercising of considerable imagination. It is hoped that the last element will be replaced by electromyography.

Progress in applying electromyography to anatomical problems has been rather slow. To date there are not more than a dozen good papers on the solution of purely anatomical problems.

Shoulder Region: Inman, Saunders and Abbott combined electromyography with other techniques in working out the functions of the shoulder joint and its muscles. The Deltoid exhibits its greatest activity after 90° during abduction and after 110° during flexion of the shoulder. Contrary to the usual teaching, Supraspinatus acts progressively with Deltoid and does not simply initiate abduction.

It was shown that Subscapularis, Infraspinatus and Teres Minor constitute a functional group that acts as the second or inferior component of a force couple. Thus, these muscles are acting continuously in both abduction and flexion.

Teres Major is not a kinetic muscle but is important in maintaining static posture of the shoulder. With an increase in load there is an increase in activity of Teres Major.

The function of Serratus Anterior, Levator Scapulae and Trapezius as rotators of the scapula on the chest wall was emphasized.

Biceps Brachii: Sullivan, Mortensen, Miles and Greene confirmed in a short paper some of the accepted teachings regarding the function of this muscle.

Trapezius: Mortensen and Wiendenbauer confirmed the usual teaching that Trapezius is active during elevation and

retraction of the shoulder girdle and during flexion and abduction of the shoulder joint. The upper 1/3 of the muscle is most active during elevation, the lower 2/3 during retraction. There is a progressive increase in activity from the cephalic to the caudal areas of the muscle during both flexion and abduction. Trapezius is most active during the latter ½ of all movements although there is a falling off of activity near the end of the movement during abduction.

Wrist: Dempster and Finerty reported a series of experiments on the relative activity of wrist-moving muscles in the static support of the wrist joint. When the forearm and hand are rotated so that gravity acts at different angles to the plane of action of a given test muscle, potentials from that muscle vary with the angular position. Typically, a muscle was most active in the direct agonist position while it was directly counteracting gravity. As an antagonist a muscle shows between 1/3 and 1/4 of its activity as an agonist.

Muscles of Mastication: Moyers' work proved that various movements of the temporomandibular articulation are effected by the regulated interaction of several muscles. Depression of the mandible is performed by Lateral Pterygoid and Digastric, although the latter is more active near the end of the movement. Elevation is achieved by the coordinated activity of Medial Pterygoid, Masseter and Temporalis. Lateral movements are brought about by the ipsilateral contraction of Temporalis and contralateral contractions of the Lateral and Medial Pterygoid muscles. Protraction is produced by Medial and Lateral Pterygoids. Retraction is effected by the middle and posterior fibres of Temporalis. The suprahyoid group of muscles appear to stabilize the mandible in all its movements. The activity of the muscles of mastication is similar in children and adults in spite of their differences in dentition.

Intrinsic Muscles of the Back and Abdomen: Erector Spinae (Sacrospinalis) shows activity during flexion or forward bending of the trunk from the erect posture but not normally on extension, according to C. E. Lewer Allen. At first this appears to be contrary to the usual teaching, but a little consideration makes this finding easily explainable. These muscles are more concerned with the function of "paying out" than those of a prime mover. In full flexion of the spine, Erector Spinae no longer acts because the ligamenta flava and other posterior spinal ligaments take the weight of the forward-bending body.

Rectus Abdominis is very active in flexing the trunk while the subject is supine but very inactive during the same movement when he is erect.

Findings similar to those of Lewer Allen were reported independently by Floyd and Silver. They also showed that Rectus Abdominis is passive during straining whereas External and Internal Obliques are chiefly involved. The patterns found in active expiration, coughing and singing were like those during straining. During ordinary standing the Internal Oblique appears to be in constant contraction, a fact that may be related to the protection of the inguinal canal.

Superficial Thigh and Hip Muscles: The chief points established by Wheatley and Jahnke for this region were the following. The Tensor Fasciae Latae is *not* a flexor but is an extensor and medial

rotator of the thigh. The muscles of the thigh act in an associated manner to produce a movement rather than as straight agonists and antagonists, especially in the hip region.

Muscles of Locomotion: Professor Arienti of Milan, using elaborate treadmill and myographic apparatus, has been working on the function of the muscles of the lower limb during walking. He shows that it is not possible to forecast the exact phase of activity of a muscle during walking by examining the muscle while it produces its prime movement. For example, if the Semitendinosus and Semimembranosus muscles are examined while the two are producing a voluntary movement, such as flexion of the knee, the two muscles are found to act synchronously and synergistically. On the other hand, when the subject is walking the Semitendinosus flexes the knee during the swinging of the limb while the Semimembranosus acts mostly during the phase when the foot is on the ground.

The two heads of Biceps Femoris act together during a free voluntary movement. However, in walking, the short head acts as a flexor during the swinging phase and the long head acts as a flexor during the phase when the foot is on the ground.

Arienti has demonstrated the same asynchrony within the heads of the Quadriceps Femoris and the Triceps Surae.

Hirschberg and Nathanson at New York University are investigating normal and abnormal gaits. Only one muscle group, the calf muscles, start to contract in the middle of the "stance phase", and this group is the most active in the normal gait. Six muscle groups, including the hip muscles, the Quadriceps, and the hamstrings, show electrical activity during the transition from swing to stance phase. Of these muscles only the Gluteus Medius continues to contract beyond the middle of the stance phase. The others cease contracting within the first 1/3 of this phase. At the transition from stance to swing, the adductors, and in some cases, the hamstrings contract. During the swing phase only the pretibial group is active. Frequently a burst of activity of the Gastrocnemius was found in the centre of the swing phase. Some of these findings have been confirmed by our own tests.

Muscles of the Leg and Foot: The leg muscles have received passing attention by electromyographers but no definite worthwhile results have appeared in the literature.

Using a Philips, mark IV, electromyograph, a highly reliable instrument evolving from Jasper's original equipment, Dr. J. F. Murray and at present Dr. Johanne Bentzon have cooperated with me in making records from the muscles of the leg and foot. Since we use unipolar needles combined with a skin electrode we are able to isolate the various closely packed muscles.

It has been possible to confirm in a general way the textbook description of the actions of these muscles with some slight modifications. Our material at present is inadequate to draw definite conclusions.

It appears that a role ascribed to Tibialis Anterior and Peronei during ordinary standing—the maintenance of the long arches of the foot—is not valid. These muscles act only intermittently and reflexly whenever balance is about to be lost while the subject is standing quietly. We are impressed by the lack

of any activity in leg muscles and even intrinsic foot muscles in many individuals during ordinary standing.

"Tone": These studies have carried us on to the consideration of *"tone"*, a word used freely by physiologists and anatomists. It has been defined in various ways but is usually meant to be based on Sherrington's "reflex postural tonus". Sherrington and his colleagues showed that a decerebrate cat, when stood up, maintains an upright posture through a reflex excitation of scattered motor units acting asynchronously in the extensor muscles. It is unfortunate that the word "tone" was then used by others to denote a similar hypothetical activity of all muscles in all mammals at all times. It has been described in skeletal muscles, even those unassociated with posture, as being due to asynchronous contractions of motor units maintaining a constant "tension" at rest. Electromyography has shown that there is no activity in human skeletal muscle at rest. The condition of "tension" accompanied by the normal "feel" of a muscle is apparently not due to neuromuscular activity but to some inherent passive elasticity of the muscle. Clemmesen has devoted a great deal of time in a recent paper presented to the Royal Society of Medicine in elaborating and proving this thesis.

Posture: Arne Lundervold, of Oslo, and Akerblom, working independently in Stockholm, have done considerable work to determine the ideal posture for several types of sedentary work. Similar work has been done at the Institute of Aviation Medicine in California by Wells and Morehouse. Preliminary papers are appearing on investigation of the ideal positions at work but time will be necessary to evaluate the results.

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PERFORATED GASTRODUODENAL ULCERATION

D. JUBB, M.D.

Introduction

Knowledge of the existence of gastroduodenal ulceration dates back at least to the days of Galen, and mention of perforated duodenal ulcers was made in the literature as early as 1688 by Muralto.[1] Since then much work has been done to gain a better understanding of the disease and its complications, and towards improvements in treatment, but as yet there are still great gaps in our knowledge. One recent innovation in the field of treatment has been the advancement of conservative therapy to supplant the established surgical approach for the complication of perforation, and it is this topic which has prompted the preparation of this paper.

Incidence

Perforation is one of the major complications of peptic ulcers, and its incidence, quite naturally, parallels in many ways that of chronic peptic ulcer. It occurs in about 12-15% of all patients with ulcers[2] with a strong predilection for males, less than 2% occurring in females,[3] and with a wide age incidence ranging from infants to elderly adults. It is commonest in the fourth and fifth decades of life with 60% of cases in adults over the age of forty years.

Classification

Clinically, patients with a perforated peptic ulcer can be classified in many ways, but a helpful method from the standpoint of diagnosis and treatment is as follows:

1. *Acute perforation*:
   - (a) onset
   - (b) stage of peritoneal irritation
   - (c) stage of toxaemia
2. *Subacute perforation*

The latter is of lesser importance and as it may be quickly considered, will be discussed first.

Subacute Perforation

Subacute or masked perforation is a small or pin-point perforation of a chronic peptic ulcer through which gastric contents leak very slowly, or which is sealed off by omentum or other adjacent structures before much leakage can occur. In this group of patients the diagnosis is often missed. The patient's complaints of a bout of atypical "ulcer" pain associated with a little localized tenderness and rigidity are most often erroneously attributed to cholecystitis, appendicitis, or renal disease. This type of case is best treated by accurate medical treatment including bed rest, bland diet, and antibiotics prophylactically. The prognosis is excellent. In most cases spontaneous healing occurs in ten days with a disappearance of the local tenderness. The main complication of these subacute perforations is the development of an abscess, either locally or in the subphrenic space, which may ultimately demand surgical drainage.

Acute Perforation

By far the more dangerous group and the patients most commonly rushed to medical attention for emergency treat-

ment are those with the acute "blow-out" type of perforation. In these cases an ulcer penetrating into the relatively free anterior or superior surface of the duodenum or the anterior surface of the stomach perforates into the peritoneal cavity or into the lesser sac. Perforation usually occurs spontaneously in a patient who has been suffering an acute exacerbation of his peptic ulcer symptoms. More rarely the disaster may be precipitated by a heavy meal, a barium enema, overexertion or a bout of indulgence in alcohol.

(a) Onset

In a typical case the perforation occurs with startling suddenness, the patient being able to give accurately to the minute the time of onset of symptoms. The attack begins with severe epigastric pain often described as a "knife-thrust" which spreads quickly over the whole abdomen and often radiates into the chest, back and shoulders. The patient immediately collapses into a state of shock becoming cold, pale, and sweaty with drawn countenance, subnormal temperature, increased pulse rate, and shallow respirations.

(b) Stage of Peritoneal Irritation

As the peritoneal cavity becomes flooded with gastric contents over the next one to two hours, the full blown picture of peritoneal irritation develops. At this stage the abdominal pain is severe but not continuous, aggravated by breathing or turning over in bed, and usually generalized over the whole abdomen though rarely it tends to remain localized to the epigastrium. Vomiting may occur but is not severe. The patient's colour usually improves, his facies becomes less distressed, his pulse stronger, and his temperature normal so that, seen at this so called "tragically dangerous latent stage", the seemingly great improvement in the general condition of the patient may throw doubt upon the diagnosis. However, examination of the abdomen reveals the true situation, for it is retracted and does not move with respiration. Superficial tenderness is absent, but there is marked deep tenderness which may be most evident in the upper abdomen. The most characteristic sign, of course, is the widespread board-like rigidity of a degree common in no other lesion. If much gas has escaped, percussion may reveal absence of liver dullness, but this sign is not very reliable. More important is the finding on X-Ray of an air bubble under the diaphragm. If present, this air bubble is almost pathognomonic of the perforation of a hollow viscus, but its absence does not preclude perforation. Leucocytosis is usually present.

(c) Stage of Toxaemia

If no treatment is effected at this stage, the condition of the patient rapidly deteriorates as he goes on to develop a paralytic ileus secondary to the diffuse peritonitis. A state of profound collapse is ultimately reached—the eyes become sunken, the skin moist and cold, the temperature subnormal and the pulse rapid and weak. Vomiting becomes more frequent. The vomitus is at first stomach contents but later becomes fecal. As dehydration becomes more marked the tongue becomes brown and dry and the patient complains of thirst. The abdominal pain becomes more continuous and may lessen in severity as the general condition of the patient deteriorates. The rigidity of the abdomen lessens and is replaced by increasing distension, but the

tenderness and absence of movement on respiration remain. Constipation becomes absolute and death usually occurs in two to four days.

Diagnosis

The diagnosis of acute perforation of a peptic ulcer is not difficult in most cases when due consideration is given to the previous history of indigestion, the history and description of the present pain, the examination of the abdomen for the characteristic rigidity, and the X-Ray evidence of gas escaping into the abdomen. It has been estimated by Maginot[5] in 1948 that 80% of such cases are diagnosed correctly. However, it is not always so easy, and especially in the aged or in the fat or flabby persons, the symptoms may be more insidious in onset and atypical and less spectacular in course with less muscular rigidity, making positive diagnosis virtually impossible. A case in point is that of Mrs. T.

This eighty-two year old white woman was admitted to hospital on September 10th, 1951, in a state of moderately severe dehydration with marked distension of her abdomen. She had had epigastric pain and discomfort after meals for some years and one year ago had a severe haematemesis, but X-Ray investigation failed to reveal the site of an ulcer at that time. Four days before admission she complained of an increase in the severity of her pain and became nauseated. After she had been on a milk diet the symptoms abated, and she felt better until the evening prior to admission when she again complained of severe epigastric pain with some nausea and vomiting. Dehydration and abdominal distension developed through the night and she was admitted the next day in very poor condition. A flat plate of the abdomen did not show any air under the diaphragm nor any distended loops of bowel. At operation the next day after the patient had been rehydrated, a massive pneumoperitoneum was found and a large perforation in the anterior wall of the duodenum half an inch from the pylorus. Generalized peritonitis was present. The perforation was covered with a piece of omentum, and as much free fluid as possible was sucked out of the abdomen before it was closed. On the third postoperative day, despite intensive supportive therapy including intravenous antibiotics, the patient died.

Differential Diagnosis

The differential diagnosis of acute perforation of a peptic ulcer involves many conditions and varies somewhat with the stage at which the patient is seen.

A. *Intra-abdominal Conditions to be Considered*

In the patients seen early, just after the onset of symptoms, the severity of the pain may suggest a *colic* of *cholecystic* or *renal* origin, or *lead poisoning*. But the absence of remissions in the pain and the associated rigidity and deep tenderness are against colic. During the stage of peritoneal irritation, *perforation of another viscus* such as an acutely inflamed *gall bladder* or a gangrenous *appendix* must be considered. A carefully taken history is of great value here, for usually the acutely inflamed gall bladder or appendix will have produced severe symptoms for several days before perforation occurs. Also, the generalized collapse which appears after perforation is rarely as profound, nor the tenderness and rigidity as generalized, as when a chronic peptic ulcer perforates. A *rup-*

tured ectopic gestation is rarely confused with a perforated ulcer although the onset may be as rapid and as severe, because irregularities can usually be found in the menstrual history, and the patient shows the characteristic pallor of a severe intraabdominal haemorrhage, with the abdominal signs more localized to the lower quadrants and with a doughiness rather than true rigidity. *Early acute pancreatitis* often gives rise to great difficulties in diagnosis and is very commonly confused with a perforated ulcer. However, diffuse cyanosis, rapid pulse, persistent vomiting and little or no true rigidity in a patient seen early in the illness are all much in favour of the diagnosis of acute pancreatitis, and if the serum amylase value is found to be elevated the diagnosis is confirmed. Also to be considered in the differential diagnosis of perforated peptic ulcer are *mesenteric embolism, mesenteric thrombosis,* and *acute strangulation of the intestine.* These cases resemble the late cases of perforated peptic ulcer when the stage of toxic ileus is beginning, since they all have the symptoms common to intestinal obstruction from any cause, namely, persistent vomiting, severe intermittent abdominal pain, progressive abdominal distension, and failure to pass flatus. History of the course of the illness, especially in regard to the timing of events, is most helpful in differentiating perforated ulcers from these other lesions, and the presence of any rigidity is highly suggestive of perforated ulcer since it is so uncommon in intestinal obstruction due to embolism, thrombosis or strangulation of a closed loop. The rare case of *gastric crisis of tabes* can usually be differentiated from perforated ulcer without much difficulty when the absence of rigidity and distension are noted along with other signs of tabes such as an Argyll Robertson pupil and the absence of knee jerks and of tendo Achilles pain on deep pressure.

B. *Extra-abdominal Conditions to be Considered*

Extra-abdominal conditions to be considered include *coronary thrombosis* and *pneumonia with pleurisy.* Careful investigation into the type and radiation of the pain, the absence of expected signs and symptoms in the abdomen such as rigidity, and the presence of signs and symptoms in the chest or cardiovascular system usually remove any doubt as to the correct diagnosis.

Surgical Treatment

The long accepted method of treatment for a perforated peptic ulcer is surgical and dates back to the first reports of successful suture of a perforated ulcer by H. Kriege in 1892.[6] The rationale behind the surgical treatment is that it is an emergency life saving procedure without which the patient would die. Therefore, manipulation is kept to a minimum and any procedure except simple closure of the perforation is condemned by most authors. One notable exception is S. S. Judin of Moscow who in 1937 published the results of a series of 426 cases treated with routine gastric resection for perforated ulcer in which his mortality rate of 8.4% compares not too unfavourably with that of a similar series of cases treated by simple closure. This, however, is unusual and the commonly recommended operation consists of opening the abdomen through a right paramedian incision, placing three interrupted catgut sutures across the perforation parallel to the long axis of the duodenum and tying them loosely over

a piece of omentum placed over the perforation. Fibrin formation between the omentum and the duodenum or stomach seals off the perforation. Any gross exudate in the peritoneal cavity is sucked out without traumatizing existing adhesions or other organs, and the abdomen closed without drainage.

Indication for Delay in Operation

Although it is classified as an emergency procedure, experience has taught the surgeons that this operation is more safely undertaken if the patient's clinical state is improved first even if it means a delay of up to eight to twelve hours after hospital admission rather than admitting the patient directly to the operating room. In this regard Dr. Roscoe Graham has set down the following criteria as sufficient to justify delaying operation to improve the general clinical condition of the patient: "(1) A severe nutritional disturbance, which can be gauged by the diet taken immediately pre-operatively; (2) severe dehydration; (3) severe anaemia; (4) a low blood pressure, rapid pulse rate, severe abdominal pain, as a part of the general picture of shock." Thus adequate intravenous fluids, heat, sedation, and oxygen are given to all patients pre-operatively, and dependent upon the views of the surgeon, the stomach is kept empty by means of a Wangensteen suction apparatus. Pre-operative use of antibiotics is also a matter of opinion as yet with some authors recommending full doses of the various drugs available and others using none at all.

Anaesthesia in the Surgery of Perforated Ulcer

The choice of anaesthesia has become recognized as an important factor in the surgical handling of a perforated ulcer case, with spinal anaesthesia being the choice at most centres because it decreases diaphragmatic activity and so lessens the tendency of the intraperitoneal exudate to disseminate. To overcome any apprehension and restlessness on the part of the patient intravenous sodium pentothal is used as a supplementary measure. In patients too ill for spinal anaesthesia, local block anaesthesia transperitoneally is recommended.

Postoperative Care

Postoperatively these patients are treated with oxygen, sufficient sedation, preferably morphine or pantopon, to control pain, continuous intravenous saline solution with glucose, any of the various antibiotic agents, and repeated or continuous aspiration of an indwelling duodenal tube. The patient is turned every two hours, encouraged to move his legs, and stimulated to overbreathe and to cough by the administration of carbon dioxide every two to four hours the first postoperative day. In a patient doing poorly, complications are searched out in the chest, subphrenic spaces, or in the pelvis, the latter by rectal examination.

Mortality Rate

The mortality rate of surgically treated cases of perforated peptic ulcer has decreased steadily over the years to reach a new low of 1.4% as reported in a recent series published by Vaughan and Warren. They attribute much of the improvement to a combination of factors including better operative technique, increased knowledge of fluid balance, better anaesthesia, ease of blood transfusion, and the advent of antibiotics and chemotherapy. Although the operation for perforated peptic ulcer is in no way intended as a cure of the chronic ulcer, a glimpse

into the long range effect of the treatment reveals that in many cases an ulcer cure is effected. In a series of cases reported by Martz and Foote it was found that most of the cases were free of ulcer symptoms after the successful treatment of their perforation, and in some cases no radiological evidence of an ulcer could be found as early as three to four weeks after operation.

Conservative Treatment

The nonoperative treatment of perforated peptic ulcer, although recognized as possible in the occasional case as early as 1870, was not considered as an acceptable method of treatment until as late as 1945. However, during the last six years it has been gathering many supporters, among them being Hermon Taylor of London, and many others.

The therapeutic measures which constitute the conservative method of treatment include the following:

(1) Indwelling duodenal tube with continuous suction or repeated aspiration every half hour, usually required for a total of 48-72 hours.

(2) Intravenous 5% glucose in saline in an amount adequate to replace the total output plus three pints extra every 24 hours.

(3) Morphine gr. 1/6 or pantopon gr. 1/3 every four to six hours for three to four doses.

(4) Chemotherapy: penicillin 40,000-60,000 units every four hours alone or with streptomycin.

(5) Deep breathing exercises.

(6) Diet of sips of water from the start increased to diluted milk as soon as the duodenal tube is out.

Response to Conservative Treatment

The first six hours is the critical period during which the patient must be observed with the utmost diligence for the signs of success or failure of the treatment. Evidences of recovery include decreasing symptoms, falling pulse rate, disappearance of rigidity and the presence of active bowel sounds. The patient's colour improves and his agonized expression changes to one of resignation. Successful aspiration of the stomach causes increased relief of pain in one to two hours, and within a few more hours the abdominal rigidity begins to diminish in extent and degree, receding from left to right iliac fossa and then up the right side to the epigastrium. General tenderness disappears in 48 hours, but local tenderness over the site of the perforation remains for 72-96 hours. Equally important are the signs of failure of the conservative method, i.e., the indications for its abandonment. The two important ones are persistent or recurrent severe abdominal pain, and any extension of the area of tenderness and rigidity. Changes in the pulse and temperature are not as reliable because they lag behind local pathological changes.

The two main problems to be reckoned with when this form of treatment is being considered for a case of perforated ulcer are the establishment of the correct diagnosis and the need for expert judgment in choosing suitable cases. The important points in diagnosis and a discussion of the differential diagnosis have been considered earlier; it remains only to add here that in those cases where the diagnosis is at all doubtful, or where a condition demanding operation cannot be definitely excluded, laparotomy is still the only acceptable form of treatment.

Contraindications for Conservative Treatment

The selection of suitable cases for conservative treatment and the recognition of situations in which it is unlikely to succeed are perhaps the most vital points in this whole method. Stead of London has suggested the following contraindications for the nonoperative treatment:

(1) No experienced surgeon available—experience is necessary to reduce the risk of error in diagnosis and for intelligent observation during the vital first six hours of treatment.

(2) When gross peritoneal contamination has occurred—the patient needs the benefit of aspiration of the irritating fluid.

(3) Cases of doubtful diagnosis.

(4) Any signs of failure of the conservative method.

To these Vaughan and Warren would add a fifth—the uncooperative patient in whom it is impossible to establish the necessary routine.

Mortality Rate

The mortality rate of patients with perforated peptic ulcers treated by the nonsurgical method varies somewhat from series to series but seems to be almost as low as for cases treated surgically when an experienced person is in charge. The advantages stressed by the supporters of this method include no postoperative shock, no postanaesthesia complications, no aggravation of existing peritonitis nor trauma to the peritoneum, duodenum or stomach, fewer postoperative chest complications and subphrenic abscesses, and no wound infections or incisional hernias. However, none of the workers with this form of treatment advocate its use indiscriminately to replace the surgical method. They suggest that it is perhaps the best method of handling the majority of cases with laparotomy being reserved for the unsuitable ones or those not responding well to the conservative regime. Vaughan and Warren being less optimistic, conclude from their work that "perforated ulcer patients should usually be operated upon but that the conservative treatment is advisable in cases in which it is certain that the perforation has healed, peritonitis is localized, or the patient is too poor a risk for surgical intervention."

Status of Conservative Treatment

Actually the status of the conservative method in the treatment of perforated peptic ulcers today is best summed up by Stead who in his article of January of this year concludes that "the conservative method is still on trial and indications for its use are not yet really established."

Thus it seems logical for me to conclude this paper by saying that, for the present at least, surgical treatment is still to be recommended as the treatment of choice unless definitely contraindicated, with the nonsurgical method acknowledged as a substitute in experienced hands.

ACKNOWLEDGMENT

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EDITORIAL

A GOOD EXAMPLE

Although the new building program of the University has aroused much interest and admiration among most medical students, the renovation of the Student Common Room of the Toronto General Hospital is something which has excited even the most apathetic returning student.

For many years, the Common Room has been a subject of debate at Medical Society meetings but improvements were postponed due to a fear of rising prices, or because it was felt a waste of money to improve an environment soon to change under the hospital's construction program. Meanwhile, students were forced to eat hasty lunches in an overcrowded, dimly lit room where conditions were often unhygienic and depressing.

However, during the past summer under the leadership of a committee from the Medical Society, a small group of willing volunteers completely refinished the Common Room and because of their liberal spending of personal time on the project, completed the work for less than half of the estimated cost. The room today has a bright cheery atmosphere which now makes it an excellent temporary structure until new accommodations are found.

It would be wise for all students using the facilities of the Common Room in the future, to speculate briefly on what can be done by a determined group of undergraduates, and to wonder if other projects could not be carried out in a similar manner.

SIR ARTHUR KEITH*

J. SALTER, 5T5

The material for this paper is taken largely from the book "An Autobiography" by Sir Arthur Keith. First, a bit of an introduction: he was born in 1866 near Aberdeen, and is now living in England in what was once Darwin's home, in the parish of Downe, near London. By study and interest he is anatomist, but this interest soon led him to an even greater man—anthropology, and studies into the antiquity of man, and his evolution. During his life he has given many lectures, and written many papers on anatomical and anthropological subjects. He is a member of the Anatomical Society, the Anthropological Society, and is now honorary member or fellow of many more. For twenty-five years he was Conservator of the Museum of the Royal College of Surgeons in London. He has, and has had, many friends —there is hardly a page in his book where he does not mention some friend or colleague. He was knighted in 1921.

Arthur Keith was born at Quarry Farm, where his father, John Keith farmed for a living. At the age of eight, Arthur and his family moved to Kinnermit, which was to remain the family home for many years. By the age of seventeen, he looked forward happily to life as a farmer, for he had not excelled himself in his early school years. However in 1822 a third year student at the University of Aberdeen came as a boarder into the Keith household. His tales of the life of a student appealed to Arthur, and he began to hope for a University education. The boarder and his friends felt there was not the slightest hope for him as a student in Arts, but it was not so as regards to Medicine. Thus he entered Gordon's College in Aberdeen to study the Greek and Latin necessary for entrance into medical school. Within two years he entered Marichal College as a student in Medicine.

Here he met a man who proved to be a great friend to him the rest of his life—Jamie Trail—the Botany Professor. He helped Keith through the leaner years as an Anatomist. Arthur Keith became interested in Anatomy, though he felt he profited more from the laboratory work, than from the lectures. Four years later he graduated from Marichal College with highest honours. He set out from the University with great hopes, and even greater ambitions.

The next few years were spent in a variety of places and positions. His first position was as an assistant at the Murray Asylum—an institution for the well-to-do insane. Keith left three months later—his diagnosis of a suicide of one of the patients was considered "the first blot on the record of the Murray" and he was dismissed by the enraged head of the institution because "his health was being undermined". Later he set out for Siam for three years, where he was the company doctor at a gold mine. Besides looking after the sick, he did numerous dissections on monkeys and apes, making careful drawings and descriptions of all

*From a paper delivered to the Osler Society, Sept. 25, 1952.

he did. This interest he continued in later years by reading all he could on the Primate Order thus compiling a book—which was never published—"A Systematization of our Knowledge of the Primate Order".

On returning to England, he spent two years in gaining his F.R.C.S. at University College in London, and his M.D. at the University of Aberdeen, for which an original thesis was required. For this thesis he submitted his chapter on the muscles in the various members of the Primate Order. The following year was one of study and waiting. He studied the rate of growth of the brain and the spinal cord in the Primates, and visited the various medical museums in London. He tried for post after post without success. He even advertised in a medical journal "Wanted by an M.D. and F.R.C.S. a medical post", but apparently no-one wanted anyone with those qualifications, for he received not a single reply. At last, at the end of a year—during which he was helped financially by Professor Trail—he became Senior Demonstrator of Anatomy in the School attached to the London Hospital. He remained at the hospital for some years, where he soon was appointed Professor of Anatomy. During these years he investigated the mechanism of breathing in man—he demonstrated the movements of the root of the lung, the movements of the ribs and diaphragm, and the importance of the pressure of the abdominal contents in respiration. He also demonstrated the sinu-auricular node, the bundle of His and related structures in the mammalian heart.

Let us now consider his work as Conservator of the Museum of the Royal College of surgeons, which position he held from 1908 to 1933. The first task he undertook was to study the history and catalogues of the Museum. In 1800 the Corporation of Surgeons became known as the College of Surgeons, and the College was given custody of the vast number of specimens collected and dissected by John Hunter. William Clift was the first Conservator—he had been the devoted helper of John Hunter, and he set about the immense task of cataloguing all the specimens in the collection. In 1827, as he was making poor progress in this task, Richard Owen was appointed to help him with the largest section in the museum—4000 specimens Hunter had collected to illustrate the manner of bodily function in all kinds of animals. Richard Owen, the next Conservator, wished to make the museum one of Comparative Anatomy. He naturally met with opposition from the surgeons, and he left to lend his services to the British Museum of Natural History. The additions made by the next Conservator, Sir William Flower, were those which would exhibit the principles of evolution. Then came Charles Stewart—more of an artist than an anatomist, and as Sir Arthur Keith says "beauty appealed to him more than function; many of his additions were things of rare beauty". Considering the history of the museum, Keith resolved that his additions must serve the cause of surgery. Here, however, he was faced with a conflict—his interests were mainly anthropological, and he really wanted to add to the museum things which would throw light on human evolution. However he managed to do both, although his addition of fossil skulls and the like was met with resistance by the Museum Committee.

Keith built up the odontological sec-

tion of the museum, and added fossil teeth to the collection. He was instrumental in preparing and collecting specimens illustrating the types of wounds received in warfare. He collected skulls—ancient and modern—in order to have a representative picture of the skull types found in the British people. He collected specimens illustrating the features found in acromegaly and other pituitary disorders. Lord Moynihan, President of the College in Keith's later years as Conservator, aided and abetted him in his interests in anthropology, and helped him to get fossil skulls and the like for the museum.

Keith's work in Anthropology would be in itself a paper. It, and his ambition, were the motivating factors in his life. He studied the fossil discoveries made in his time, and the earlier ones, and his knowledge of anatomy helped him to interpret the importance of the discoveries. He studied Java man, Piltdown man, and other fossil skulls. Perhaps his greatest contribution was his hormone theory of evolution. He felt that hormones provided a mechanism for human evolution. For example, the features seen in acromegaly are very similar to those seen in Neanderthal man. Keith feels that with the isolation of various groups, certain hormonal abnormalities and differences would tend to be perpetuated through generations, thus certain characters would develop. He feels that hormones might explain the development of different races of mankind.

In conclusion, Sir Arthur Keith is a man of research, study and thought. He has been interested in all ramifications of anatomy. In Anthropology he has sought to light up the few visible spots on the long road of human evolution, and tried to fill in the complete courses of that road. He has not formulated theories, or made discoveries that have led to radical change in the direction of human thought on scientific problems. But he has helped, as did and do other men like him, to illuminate the pieces which, some day, will form a coherent picture.

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IDIOPATHIC ULCERATIVE COLITIS

JAMES K. MURRAY, 5T3

Idiopathic ulcerative colitis is a non-specific inflammatory lesion of the colon and rectum occasionally involving the distal ileum.

Etiology

There are five groups of factors which have been held responsible for this disease.

(1) *Bacterial and Viral.* For many years the Bargen diplococcus attracted considerable attention. It is now felt that this is a pleomorphic streptococcus rather than a specific bacterium. Antibiotics do not conclusively alter the course of the disease and microorganisms therefore would appear to be secondary rather than primary factors.

(2) *Allergy.* Many cases are sensitive to milk and do better on a milk free diet. It is unlikely however, that this is a primary factor.

(3) *Nutritional Deficiency.* Many cases excrete large amounts of thiamine in the urine. Dennis at the University of Minnesota suggests giving large amounts of thiamine to these cases.

(4) *Excessive Lysozyme Production.* Florey in 1933 postulated that organisms were prevented from invading the gut wall by a layer of mucus. In this case an enzyme causing its removal might be a factor in producing the ulceration. Although an abnormal amount of lysozyme is found in the stools in this disease, lysozyme is also found in pus cells and its content in the stools varies directly with the number of pus cells. Recent work indicates another mucinase not associated with pus cells or bacteria is present in the stools of patients with chronic ulcerative colitis.

(5) *Psychosomatic Factors.* Acute exacerbations of idiopathic ulcerative colitis occur in periods of stressful life situations. It is in the person who is over-dependent on the approval of others or who suffers extreme degrees of frustration, humiliation, anger and resentment, that we find this disease. These people submerge their personal feelings, and external calm conceals a seething cauldron of emotion. Often these are feelings of guilt or unworthiness in relation to parents or spouses.

Pathology

The picture is one of non-specific inflammation. The colon is involved either partially or completely during the initial episode. Usually the rectosigmoid and occasionally the terminal ileum may be involved. Early in the disease the mucosa is hyperemic, glistening and edematous and may be covered with an inflammatory exudate. The ulcers may be arranged along the taenia coli and often coalesce. Between areas of ulceration polypoid hyperplasia may occur. The submucosa and muscular layer are edematous in the early stages and later undergo fibrosis. Contraction of the fibrous tissue may cause marked narrowing of the lumen. The mucosa may be so denuded that it is very susceptible to perforation and the mesenteric lymph nodes may be enlarged and inflamed.

The microscopic appearance is that of a chronic bacillary dysentery. At the ulcer site the mucosa has disappeared, the submucosa is thickened and infiltrated with round cells and the peritoneum over the deep ulcers is thickened.

Clinical Picture

Local. If the onset is gradual, the first abnormality noted may be the presence of blood and mucus in the stool. The stools become softer and occur with greater frequency and urgency, finally becoming liquid. They contain fecal material, serous exudate, blood, pus, mucus and gas while defecation may be associated with tenesmus.

If the onset is sudden, loose watery stools containing blood, gas and mucus may be passed almost continuously. Abdominal cramps preceding and relieved by defecation occur in both types and rectal soreness may be annoying.

General. Anemia, dehydration, prostration and loss of weight are frequent findings. The temperature curve is of the intermittent or remittent type. Acute arthritis, tachycardia and erythema nodosum may also be present. Tenderness to palpation over sigmoid and cecum is common.

Laboratory Findings

(1) Hypochronic normocytic or microcytic anemia depending on blood loss and degree of iron deficiency.

(2) Leucocyte count normal or elevated.

(3) Prothrombin time occasionally elevated.

(4) Base deficit may be present as shown by the low sum of chloride and bicarbonate.

(5) Alteration in A/G ratio.

Sigmoidoscopic Picture

The earliest detectable change is minute petechiae in a slightly edematous mucosa which later becomes hyperemic and glistening with an excess of mucus. Swabbing reveals a granular surface which bleeds easily. Ulcerations are of varying sizes, but may be covered due to the edema. In later stages there is little or no normal mucosa and large amounts of thick mucopurulent exudate cover the ulcers. Due to the extreme friability of the gut wall, sigmoidoscopy in acute cases should be delayed until pain spasm and irritability have disappeared. In the quiescent chronic stage the surface is pale and firm, does not bleed easily and pseudo polyps may be visible.

Roentgenologic Picture of Colon

Characteristic changes depend on the site and extent of the lesion and the degree of spasm. Early and characteristic are the saw tooth borders of the barium filled segment. With extensive mucosal destruction the normal pattern is lacking. Changes are best demonstrated by the air-barium contrast technique, the haustral markings are absent, the colon is shortened and the lumen narrowed. It is the "lead pipe" colon. Irregularly placed filling defects indicate polypoid hyperplasia. Terminal ileum if involved is rigid and dilated without normal markings.

Differential Diagnosis

(1) Amebic dysentery—sigmoidoscope shows discrete irregular undermining ulcers with normal mucosa between. Stools contain Entamoeba Histolytica and response to amebicidal drugs is good.

(2) Bacillary dysentery. Differential is done on stool culture.

(3) Sigmoiditis of lymphogranuloma venereum. A history of previous inguinal lymphadenopathy in a woman, rectal stricture late in disease, hyperglobulinemia and a positive Frei test indicate this disease.

(4) Primary intestinal tuberculosis or more commonly secondary to pulmonary tuberculosis.

(5) Regional enteritis. There is a milder clinical case, absence of gross blood in the stools, and a normal sigmoidoscopic and roentgenologic picture.

(6) Diverticulitis—Readily differentiated by X-ray.

(7) Others such as uremic ulceration and malignant lesions are readily differentiated.

Complications

Local. Stricture, obstruction, hemorrhage, perforation with general or local peritonitis, perianal or rectal abscesses or fistulae.

General. Acute arthritis, erythema nodosum, thrombosis of veins of extremities, retarded sexual and physical development, amenorrhea, severe acidosis with cerebral damage due to hemorrhage into brain and manifestations of mineral deficiency. Carcinoma of a very malignant type is much more common in patients with chronic ulcerative colitis than in the population as a whole.

Course

The disease is characterized by remissions and relapses which may be prolonged if foods to which gastro-intestinal allergy exists are ingested or if the psychiatric aspects of the case are not adequately handled.

Treatment

Medical (1) *Psychogenic:* Ross and others report that these patients have a similar basic psychological structure. There are profound psychoneurotic, hysterical depressive and compulsive manifestations with psychotic characteristics often lying just below the surface. Although they are apparently pleasant and co-operative, a powerful hostility and a marked dependence on a central figure are almost invariably found. In 50-75% of cases the onset of the exacerbation can be traced to death or derangement of this figure.

Psychotherapy takes the form of sympathetic non-critical support, in which the therapist takes the role of a kindly parent. Sympathy, reassurance, massage, simple feeding routines, warm baths and symptomatic medications are used to advantage.

After the acute manifestations subside the patient is re-educated to dependence on a more normally related person. The only reliable guide to therapy is the increase or decrease in somatic symptoms.

(2) *Diet:* The diet should be milk free, high caloric, high protein, low fat, low residue, adequate in minerals and vitamins and free of gas forming and irritating foods. In acute exacerbations intravenous feeding may be necessary.

(3) *ACTH and Cortisone:* In one series of fifteen cases treated with ACTH, nine patients improved objectively and six felt better although their diarrhea was not altered. There was a rapid decrease in the inflammatory reaction. Soon after the ACTH was stopped there was a 75% recurrence but in other cases remissions of 5, 6, and 12 months were obtained. Weight loss due to adrenal stimulation and a resulting negative

nitrogen balance occurred in 50% of cases.

(4) *Antibiotics* are of use in combatting secondary infection, but are of no proven value in altering the primary disease process. Any antibiotic used should have properties free of toxic effects due to the necessity for long usage.

(5) *Acid Base Imbalance* and calcium and vitamin deficiency should be promptly corrected. Anemia is best treated by transfusion of whole blood and administration of iron in a form which does not irritate the G.I. tract. Small doses of opium may be used to relieve abdominal cramps and diarrhea for a few days.

Surgical Measures

Surgery is indicated if there is

(1) intractability on medical treatment with incapacity persisting for three months or more each year.

(2) Obstruction of colon due to cicatrization.

(3) Subacute perforation, sinuses, abscesses or fistulae.

(4) Persisting hemorrhage.

(5) Infectious arthritis.

(6) Polypoid change.

(7) Carcinomatous change.

Methods

(1) Ileostomy—permanent or temporary.

(2) Colectomy—total or partial if disease is segmental.

(3) Experimental work on dogs has indicated that a condition similar to ulcerative colitis may be produced by division of the sympathetic nerves surrounding the blood vessels in the mesentery and colon. Transthoracic vagotomy has therefore been advocated. In one series of 41 cases results were good in 16 cases, 6 were "cured" and 3 died.

It is difficult to assess the value of any type of therapy due to the remissions and exacerbations which are characteristic of the disease. Any conclusions regarding the value of any specific therapy must therefore be modified by this fact.

Prognosis

If no treatment is given the prognosis is most distressing, 50% are dead in 5 years, 76% are dead in 10 years, 15% get cirrhosis of the liver, and in acute cases acute hepatitis. The incidence of carcinoma of the colon is extremely high in this disease. The mortality rate of ileostomy varies from 5 to 25%, depending on the stage of the disease when treated. Induction of a permanent remission without resort to surgical treatment relies on the ability of the physician to discover and handle the patient's emotional problems successfully.

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FOOD POISONING

DENNIS FRASER, 5T3

The thin veneer of security embodied in community sanitation is maintained by constant and conscientious laboratory control, regular supervision and inspection, and by continual strengthening and enforcement of public health regulations.

Despite the provisions made, statutes merely maintain their status of paper inscriptions, for the odd city in North America is still to be found, vehemently opposed to chlorination of its water supply as a safeguard. And smaller rural settlements and unorganized territories have water supplies that are potentially unsafe.

Strange that the hazards of leptospirosis, brucellosis, tularemia, poliomyelitis, cholera, typhoid and paratyphoid fevers and the dysenteries should be accepted as chance hazards by communities in a land where water is served at every meal, and uncooked salads and raw sea foods are much esteemed by many.

There is still much strength in opposition to compulsory pasteurization of milk, although cholera, typhoid and paratyphoid fevers, dysentery, tuberculosis, scarlet fever, septic throat, diphtheria, salmonella, brucellosis, staphylococcal food-poisoning and poliomyelitis have been proven to have been disseminated in unpasteurized milk, and in spite of the fact that there is no record of properly pasteurized milk or milk products not subsequently reinfected having caused these conditions.

Milk products have, to date, largely escaped the application of pasteurization laws, although M. tuberculosis, staphylococci, S. typhi and other Salmonellae find these media a satisfactory source of nourishmtnt.

Sewage and garbage disposal bear direct relationship to the rat and fly populace. The proliferation of rodents is in direct ratio to the incidence of Salmonella infections and leptospirosis, while flies encourage typhoid and paratyphoid fevers, poliomyelitis, bacillary dysentery and Salmonela infections.

A. Salmonella Food Infection

Bacteriology:

S. aertrycke (S. typhi murium)—most common in man.

S. enteriditis.

S. paratyphosum B.

S. choerasuis (B. suipestifer).

Incidence: The true incidence is obscured by the great difficulty in isolating Salmonellae in the peccant food or in the faeces of victims. The incidence is again biased by the ascription of food poisoning outbreaks to Salmonella, when no detectable organisms were present.

Modes of Conveyance:

Meat, milk or eggs of infected animals.
Pollution of food by human carriers.
Pollution of food by rats and mice.

Reservoirs:

Human carriers—healthy
—mild clinical cases
—clinical cases
—convalescent (up to 18 weeks after infection)

Animal (meats passed fit for human consumption)

Horse	0.2 per cent infection
Cattle	0.4 per cent infection
Pigs	0.67 per cent infection
Sheep	0.0 per cent infection
Chickens	0.67 per cent infection—probably largest reservoir of infection by comparison of animal populace.
Ducks	1.2 per cent infection
Turkeys	2.5 per cent infection

Fifty per cent of organisms were S. typhimurium—most common in fowl.

Most outbreaks have originated from emergency slaughtered or casualty animals, and further, whenever a discovery of human Salmonella infection was made, (in the United States in one survey in 1950) fecal specimens of domestic animals for a radius of one block were tested, and in fifty per cent of cases there was a Salmonella excreter in one or more of the animals in the area.

Clinical Manifestations are of Three Types

1. Acute G.I. irritation.
2. Typhoidal syndrome.
3. Blood stream infection.

The severity and nature of the illness is somewhat governed by the type of Salmonella involved, its pathogenicity, and invasiveness. The infectivity rate of those eating incriminated food is usually high.

Incubation Period: 12 to 36 hrs.

Characteristically: sudden vomiting, diarrhoea and abdominal pain, fever and prostration. The symptoms subside in a few days.

Mortality Rate: 1 to 2 per cent.

A THEORY PUT FORWARD BY SAVAGE:

"In some cases, living infective bacilli are ingested in the food with none, or little of the toxic products. The incubation period will be therefore long, before sufficient toxins can manifest symptoms.

"In other cases bacteria are killed in the process of preparation leaving heat resistant toxins in a preformed state which can exert other effects rapidly, within 2-4 hrs. with illness 24-36 hrs., negative stool cultures and a low fatality rate".

However lab investigations of heat killed cultures of a wide variety, and ingested by animals and man provide results which repudiate the validity of the toxic syndrome hypothesis. And indeed it is believed that staphylococcal enterotoxin is responsible for the toxin type of food poisoning, as is to be pointed out later.

B. SHIGELLA FOOD INFECTION (Bacillary Dysentery)

Bacteriology:

Sh. dysenteriae
Sh. schmitzii
Sh. flexneri
Sh. sonnei

Shigella organisms are rarely associated with animal infections, but the few which do occur spontaneously in monkeys, dogs, fowl, geese, chickens and canaries are pathogenic for man.

Modes of Conveyance:

Primarily, contamination of food by carriers and flies. The consumption of flesh, milk and eggs of infected animals is not a means of human infection accounting for a lower incidence of food poisoning—though delayed recognition of Shigella food poisoning may be a factor in this low incidence.

Reservoirs:

Human: acute case
carrier (usually chronic cases)
convalescent—up to ten months — (longer than Salmonella)

Clinical Manifestations:

Acute and severe—"Bloody Flux"
Gastro-enteritis
Simple diarrhoea
Healthy carrier

Incubation Period:

1 to 6 days—average 48 hours.

Characteristically: Diarrhoea, abdominal pain, fever and prostration, severe tenesmus and bloody stools. Vomiting is less characteristic.

Fatality Rate—1 to 2 per cent.

An increase in latter years is due to a real increase caused by Shigella sonnei infections which account for ninety per cent of cases. The remaining ten per cent is due to Shigella flexner. Nurseries and Mental Hospitals are the most frequent sites of epidemics. Shigella dysenteriae produces an exotoxin responsible for mortality rates as high as twenty per cent.

C. Staphylococcal Food Poisoning

Having the *commonest incidence*, the actual incidence of staphylococcal food poisoning is unknown, since outbreaks are only reported when large groups are involved and the organism, as will be pointed out later, may be masked in culture by other bacteria.

Bacteriology—Staphylococcus Aureus.

Modes of Conveyance:

Milk and its products are the most important vehicles for food poisoning of this type, due to the considerable handling in preparation, the suitability of the media (*without signs of spoilage*), and the public desire to see pastries displayed in warm shop windows.

Ham is probably the second important mode of infection, and indeed, the only fatal case recorded in Canada was due to ham containing the enterotoxin.

Human carriers—healthy or infected —harbouring the organism in the nasopharynx is another important site.

Another important source is infected dairy cattle with or without signs of mastitis.

Clinical Manifestations:

Symptoms within 1 to 4 hrs. Nausea for a few minutes to 1 hour. Sudden vomiting, with bouts of vomiting in the next few hours. Abdominal cramps and marked diarrhoea.

Rapid, (thready) pulse.

Temperature raised—collapse apprehensiveness, numb extremities, pallor and cold sweats may ensue.

Improvement in 3 to 5 hrs., with return of color, pulse and desire for food and drink.

Anorexia, nausea, diarrhoea, abdominal discomfort and considerable loss of weight may persist for a day or two.

Mortality rate: low.

Enterotoxin Production:

The action of the enterotoxin is not directly on the G.I. system, but rather on the Central Nervous System. Food contaminated by enterotoxigenic staphylococci, can be exposed to warm temperatures or intermittently refrigerated and produce the toxin. The organism may elaborate sufficient entertoxin to render the food poisonous, at room tem-

perature within ten hours, or in a room at 85 degrees F. in five hours.

D. Botulism

Botulism is the rarest type of poisoning, but food shortages and high costs of living are motivators toward a revival in home canning and bottling—the accepted media for this organism.

Bacteriology: Clostridium botulinum.

Incidence: A recorded total of 3 Canada, or better methods of preservation in the United States, but rather an uneven distribution of the organism. In Great Britain only two outbreaks and two single cases were reported in the similar period.

Modes of Conveyance:

Hams, smoked meats and canned foods.

Clinical Manifestations: A preformed toxin—symptoms within 24 hours.

Primarily non-gastrointestinal. A general weakness and lassitude. Diplopia and ocular paralysis, pharyngeal paralysis, and laryngeal paralysis. Diaphragmatic paralysis, occasional vomiting—no fever, no headache, no pain, and full consciousness.

Death intervenes with respiratory or cardiac failure within one to two weeks. It is then, a progressive poisoning without remissions.

Other Reported Types of Bacterial Food Poisoning

B. proteus, B. coli and the green producing streptococci have all been incriminated in various outbreaks of food poisoning. Conflicting reports of investigators led to specific investigations under rigid controls by a number of workers and none of the organisms was found to produce the symptoms of food poisoning when they were given as filtrates.

Live cultures of the green producing streptococci provoked abdominal distress and diarrhoea with general malaise, and, with the fact that the incubation period before symptoms appeared was usually 12 to 15 hours, strepococcal food poisoning is probably due to infection *with living organisms* (which could be controlled by proper cooking), *and not to the action of pre-formed toxins.*

In most instances of poisoning with the above mentioned bacteria, the symptoms occur within a very few hours and even within thirty minutes, *pointing significantly to the presence of a pre-formed toxin*!

Staphylococcal Enterotoxin is *the only agent* proven beyond question to produce *a toxin type* of food poisoning! The food stuffs, implicated in the cases above, are similar to those involved in staphylococcal poisoning, and staphylococcal organisms were often present in small numbers in the poisoning food. We may then suspect these numbers to be the few survivors of myriad predecessors, which is in agreement with the theory that there is a marked decline in the staphylococcal population after early

	Incubation	Vomiting	Diarrhoea	Abdominal Pain	Fever	Prostration
Salmonella	12-36 hrs.	++	++	++	+	+
Shigella	1-6 days (48 hrs.)	±	++	++	+	+
Staphlococcus	1-4 hrs.	++++	+++	++	+	+
Botulism	in 24 hrs.	weakness, paralysis		−	−	+

phases of rapid proliferation with maximal enterotoxin production. This theory was subsequently proven valid by investigations by Dolman and others.

Again in lieu of foregoing statements regarding toxin types of Salmonella poisoning, the hypothesis can be stretched to include those Salmonella outbreaks in which occasional cases have a very brief incubation period.

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ANNOUNCEMENT OF VAN METER PRIZE AWARD

The American Goiter Association again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association, which will be held in Chicago, Illinois, May 7, 8 and 9, 1953, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or reesarch investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten double spaced copy in duplicate sent to the Corresponding Secretary, Dr. George C. Shivers, 100 East Saint Vrain Street, Colorado Springs, Colorado, not later than February 15, 1953. The committee who will review the manuscripts is composed of men well qualified to judge the merits of the competing essays.

A place will be reserved on the program of the annual meeting for the presentation of the Prize Award Essay by the author, if it is possible for him to attend. The essay will be published in the annual Proceedings of the Association.

ABSTRACTS

Differential Diagnosis of 409 Revised Cases of Acute Poliomyelitis: Dawson, K. E., La Bocetta, A. C. et al. Jour. of Pediatrics, Vol. 40, No. 1.

A classification of the disease according to the poliomyelitis-like symptoms is presented. In each case the differential diagnosis is discussed briefly.

The diagnosis of acute anterior poliomyelitis is, at best, only presumptive. The diagnosis is based on:—

1. headache
2. fever
3. increased nuchal resistance
4. spasm of the back and hamstring muscles
5. spinal fluid pleocytoses and-or lower motor neurone paralysis

Of 1420 patients admitted with a diagnosis of acute poliomyelitis 1011 were confirmed and 409 revised. The revised diagnoses included:

1. Central nervous system diseases	113
2. Peripheral nerve disease	8
3. Psychogenic disorders	13
4. Respiratory infections	114
5. Musculoskeletal disease	77
6. Gastrointestinal disease	17
7. Genito urinary disease	18
8. Diseases of skin and allied states	10
9. Acute and febrile disease	5

The diagnosis is usually one of exclusion. Spinal fluid should be examined in every patient who shows acute disease of the central nervous system. Routine blood-virus studies should be performed in every patient with a spinal fluid pleocytosis who does not have a flaccid paralysis.

N. B. Abbey

Control of Bone Length: Blount, W. P. and Zeier. J.A.M.A., Vol. 148, No. 6, p. 451.

The authors have used either symmetrical or asymmetrical placing of staples across the epiphyseal plate to control length or to correct angular deformity in limbs of 90 patients in ages from 5 to 15 years. They preferred a lower limit of 8 years, because below this age the cartilaginous nature of the epiphysis made it difficult to insert staples. Technical difficulties encountered in the method included bending and pulling out of staples, breaking of staples and cutting through the epiphysis.

They have found smooth, inert stainless steel staples of 3/32 inch diameter of specified softness most suitable. The periosteum is not cut if staples are to be removed. Three or four staples are accurately placed as a unit on either side of the epiphysis depending on the deformity to be corrected.

Since a growth spurt occurs up to two years after removal of the staples angular deformities are best over-corrected to prevent recurrence. However linear over-correction should not be done, for unless a stimulating growth factor is still present, the stapled epiphysis on removal of the staples closes several months earlier than normal.

The growing epiphysis of a child exerts a tremendous force on the staples but dense bone deposited around the staples usually prevents their cutting through bone.

C. Blazic

CLASS OF '52 INTERNSHIPS

Allan, G. W.	Hamilton General Hospital, Hamilton, Ont.
Altman, S. I.	Mount Sinai Hospital, Cleveland, Ohio
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Bow, L. W.	Toronto General Hospital
Box, D. H.	Kitchener-Waterloo Hospital, Kitchener, Ont.
Box, T. R. H.	Toronto General Hospital
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Brown, R. F.	St. Michael's Hospital, Toronto
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Evans, J. R.	Toronto General Hospital

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Rotenberg, M.	Brooklyn Jewish Hospital, Brooklyn, N.Y.
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Skorey, G. R.	St. Elizabeth Hospital, Youngstown, Ohio
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Streight, N. E.	Royal Jubilee Hospital, Victoria, B.C.

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Woollacott, P. J. V.	Vancouver General Hospital, Vancouver, B.C.
Wynne, J. B.	Toronto Western Hospital.
Zatelyn, W.	St. Joseph's Hospital, Toronto.

In an attempt to increase student participation in the Medical Journal, an award of $50 will be made to the author of the best undergraduate article submitted for publication during the present year.

Articles should be typewritten, double spaced, and approximately 300 words in length. All years are eligible for this award, and entries should be addressed to the Editor, Room 107, Anatomy Building.

NEWS AND NOTES

"The newly re-organized Medical Debating Society will sponsor a program of noon-hour and interfaculty debates for '52-'53. Anyone interested in taking part in this program is advised to call the Chairman, Miss Frieda McQueen at MIdway 1015, or the Program Director, Mr. Lawlor Loudon at RAndolph 4870, for further information."

* * *

The Donald C. Balfour Lecture in Surgery was delivered on Monday, September 29th, 1952, at 5.00 p.m. in Convocation Hall, University of Toronto. The guest lecturer was Mr. Clifford Morson, O.B.E., F.R.C.S., Consulting Urologist, National Health Service and Chairman of the Academic Board, Institute of Urology, University of London. His subject was "Ill Health and History."

* * *

The annual meeting of the Association of Canadian Medical Colleges was held on October 1, 1952, with Dr. J. A. MacFarlane presiding as president.
presiding as president.

* * *

During the past summer a group of students undertook to modernize the Students' Common Room in the Toronto General Hospital. The room has been completely redecorated and several new tables have been purchased. One of the highlights of the room is a group of paintings by well-known artists depicting scenes from Canadian life. These pictures are the results of combined donations from the Medical Arts and Letters Society and Fisher and Burpe Instrument Company. The committee responsible for this fine piece of work are: F. B. Fallis, 5T3, (Chairman); D. Vince, 5T4; P. Blundell, 5T4; S. Louden, 5T6. Assisting the committee were: F. Boughen, C. Blazic, D. Barr and R. Proud; all of 5T3. Congratulations are certainly due to all concerned.

* * *

The Freshman Reception for the new class of 5T8 was held on October 1st at 7.30 p.m. in the Women's Union. Dr. R. F. Farquharson, Professor and Head of the Department of Medicine, was the speaker and he gave the new students a hearty welcome on behalf of the Faculty. The master of ceremonies was W. T. Shaw, 5T5.

* * *

The CAMSI Art Salon was held from October 13th–October 18th at Laval University, Quebec City. The exhibits were divided into the following categories: Fine Arts, Monochrome Photography and Miscellaneous Exhibits including Color Photographs and Drawings.

* * *

During last year's transportation strike, the Medical Reading Room adopted new hours. These proved so successful that they are being continued this year. The Reading Room will now be open from 8.45 a.m.–10 p.m. on week days and 8.45 a.m.–5 p.m. on Saturdays.

* * *

The Medical Women's Undergraduate Association held its annual initiation October 14th. The speaker was Dr. E. Stevenson of Willowdale.

* * *

A new system of grading has been announced by the Faculty as follows: Honours (A) 80-100%; Pass (B) 70-79%; (C) 60-69%; (D) 50-59%; (E) Below 50%. The minimum pass standard will be 60% for all years.

* * *

The Medical Arts and Letters Society

The Medical Arts and Letters Society is an organization intended to coordinate all of those aspects of the medical student's extracurricular life which have to do with

the arts and the humanitics . . . those aspects of his student life beyond the strictly medical. All students in the faculty of Medicine are warmly invited to take part in any of the groups existing under the A & L Society, as described below. These activities are sustained by your fees, through the Medical Society.

For any information call Stan Greben. MA. 9573.

* * *

The Osler Society

This organization is for studcnts and staff interested in the humanities and history of medicine. Monthly meetings on the fourth Thursday of each month are held in members' homes, when papers are given by two members of the Society. All members are expected at some time in their course to present at lcast one paper.

Those interested in attending the next mecting please call Doug Abbey. MI. 7319.

Medical Music Society

For students interested in music, from jazz to the classics, meetings are held monthly in members' homes. At each meeting a student member or guest will present a program of recorded music.

For information call Dorothy Lieberman. LA. 0179.

* * *

Medical Photography Society

For all Medical students interested in photography. Speakers at monthly meetings will be accomplished photographers who will be speaking both to the experienced photographers and the completely inexperienced with only an interest in the subject.

All interested please call John Stears. PR. 4974.

W. Kokeny.
J. Goodwin.

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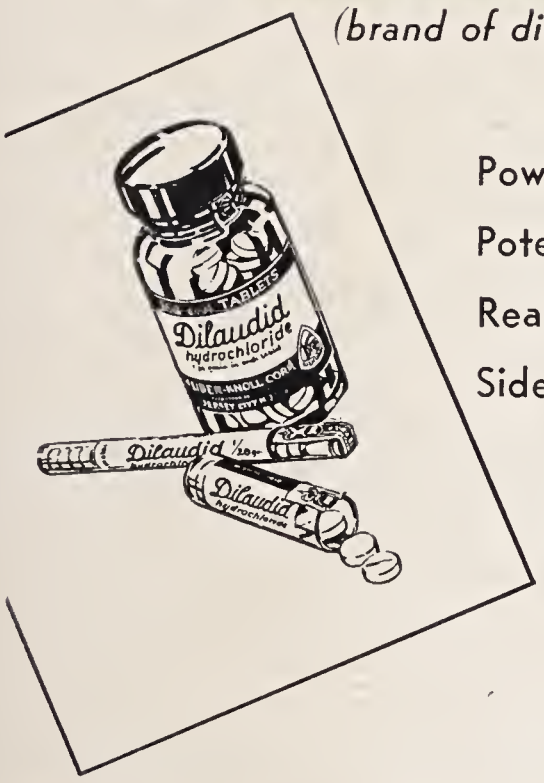

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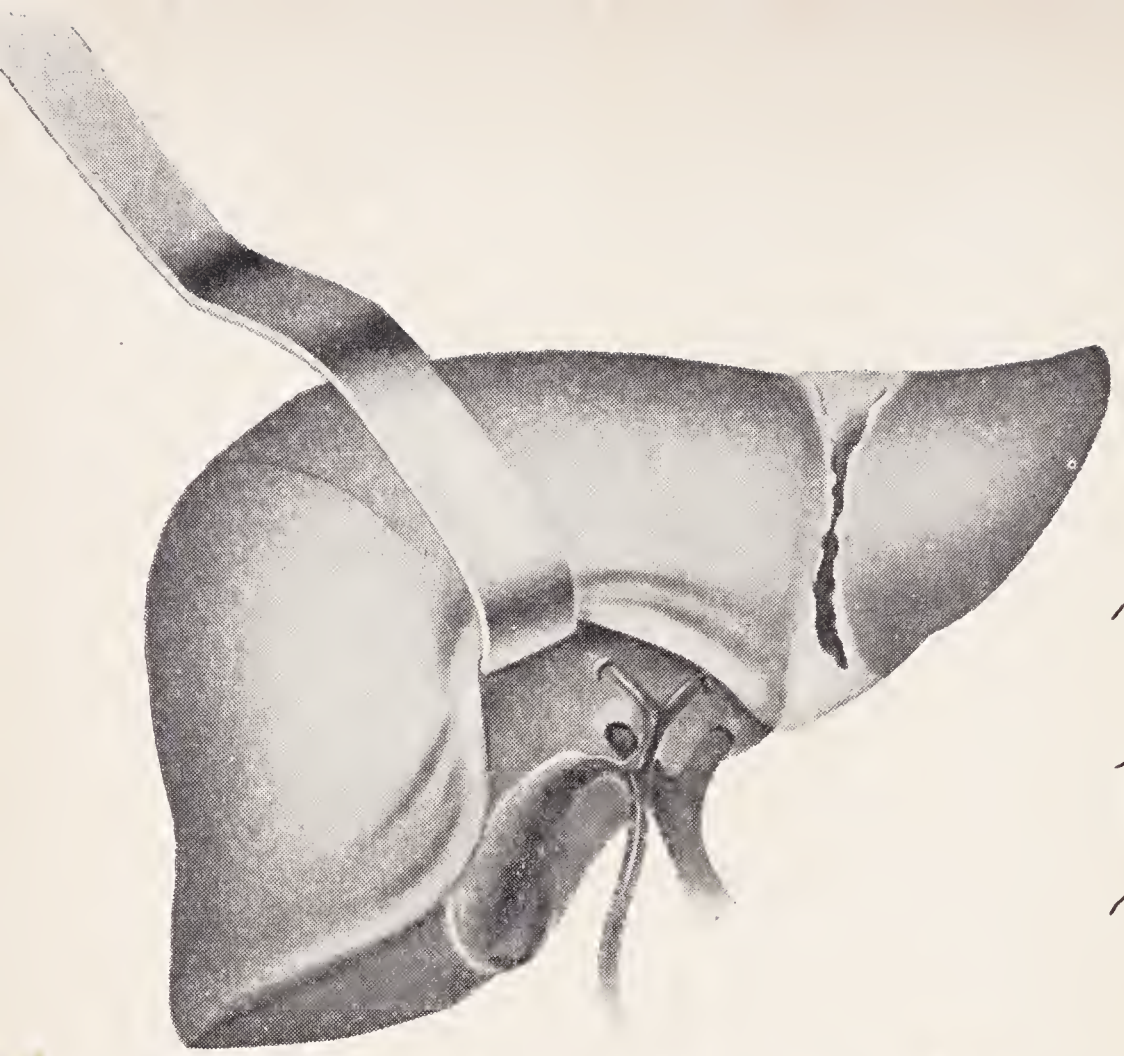

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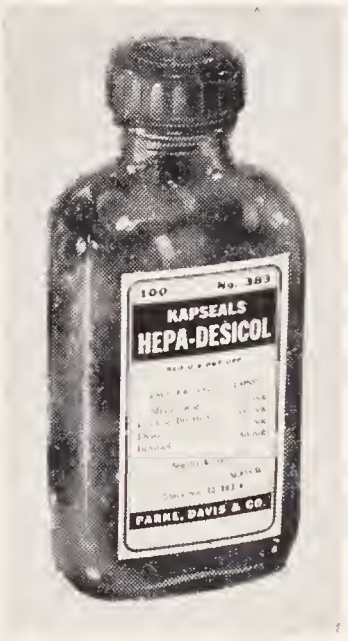

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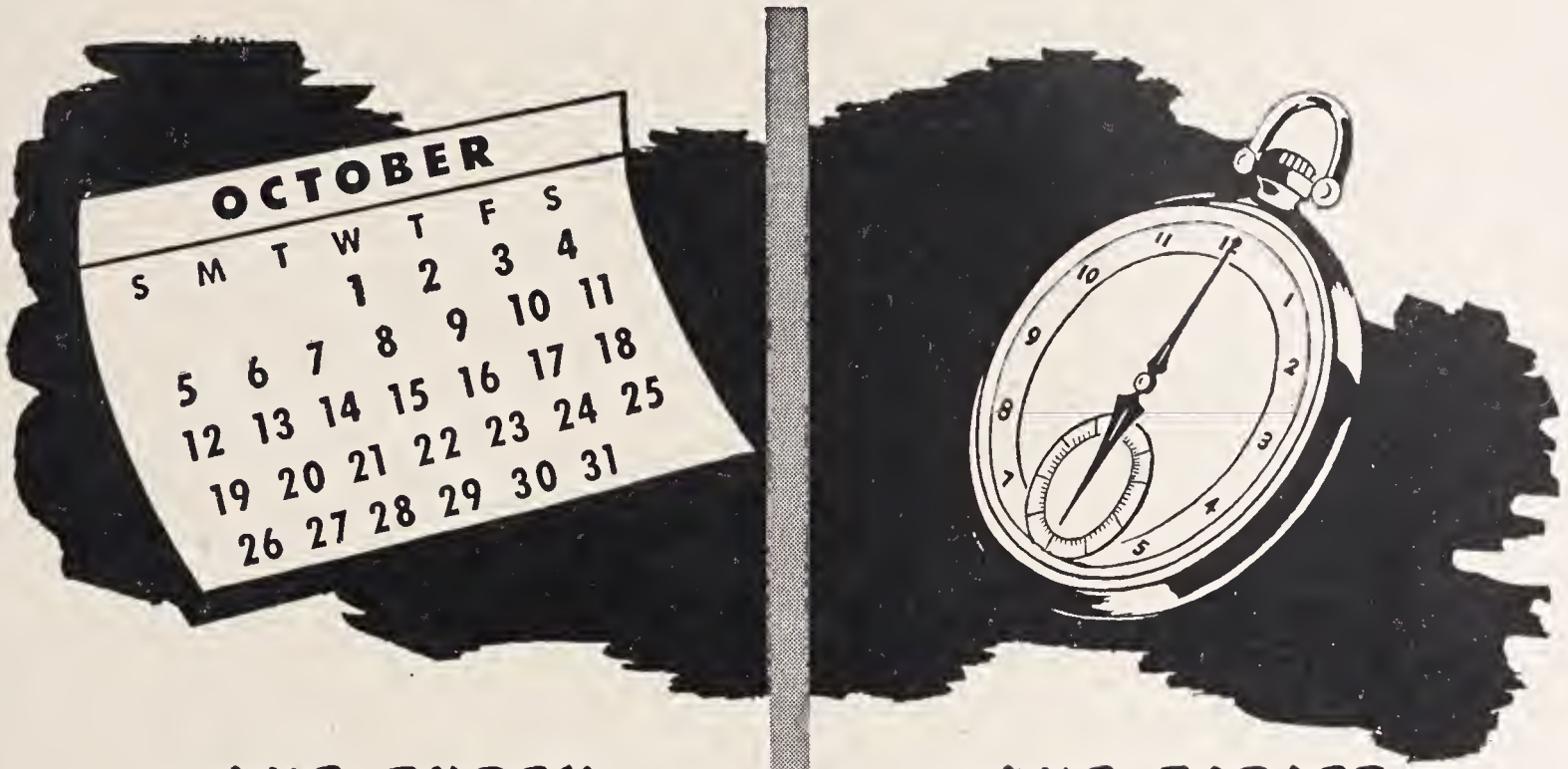

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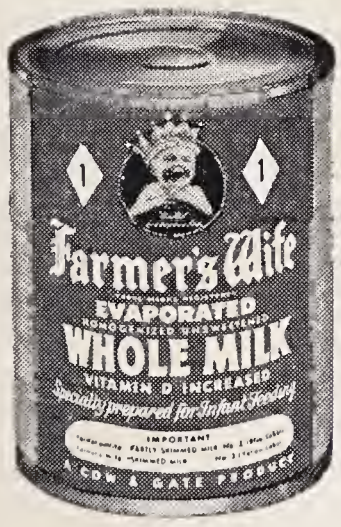
With these three types of "FARMER'S WIFE" milks you have a formula choice which simplifies the actual home preparation—insures more exact and constant milk formulae. All "FARMER'S WIFE" is Vitamin D increased to 480 International Units per pint, by the addition of pure Crystalline Vitamin D_3.

No. 1 Red Label—*Whole Milk. Butterfat 8%; Calories 45 per oz.*



No. 2 Blue Label—*Partly Skimmed Milk. Butterfat 4%; Calories 32 per oz.*

No. 3 Yellow Label—*Skimmed Milk. Butterfat 2%; Calories 29 per oz.*

● *Please write for professional information*

COW & GATE
(CANADA) LIMITED
GANANOQUE, ONTARIO

1-2B

effective, well-tolerated, esthetically pleasing

a two-year clinical study of a vaginal gel used without a diaphragm

CLINICAL REPORT* 704 PATIENTS NORTH DAKOTA

effective "This study indicates that this preparation is more effective than any other method we know.* * * During the two-year study of this Gel, conception was not effectively controlled in only 1.8% of the 704 patients who claimed regular use."

well-tolerated "All biopsy specimens, whether at six weeks, nine months, or sixteen months after the use of the Gel, show perfectly normal human vaginal mucosa . . . It would be difficult to find more nearly normal human vaginal biopsies."

esthetically pleasing "The enthusiasm with which this new method is received has convinced us that it is esthetically acceptable. The points: simplicity, ease of administration, convenience and satisfaction were expressed so emphatically and repeatedly by users that we are inclined to accept these judgments."

for simple, effective control
well-tolerated, esthetically pleasing

Preceptin
VAGINAL GEL
buffered at pH 4.5 — for optimal tolerance

PRECEPTIN vaginal gel — a major advance in conception control developed by Ortho Research Laboratories.

COMPOSITION: PRECEPTIN vaginal gel contains the active spermicidal agents p-Diisobutylphenoxypolyethoxyethanol and ricinoleic acid in a synthetic Gel base buffered at pH 4.5.

Ortho

* Hunter, G. Wilson; Darner, C. B., and Gillam, J. S.: A method of Contraception Without Diaphragm — A Two- Year Investigation. Ann. New York Acad. Sc. **54**:825, 1952.

ORTHO PHARMACEUTICAL CORPORATION (CANADA) LIMITED TORONTO
Manufacturers of Ortho-Gynol, Ortho Creme, Ortho Kit, Ortho Diaphragm.

In the Menopause...

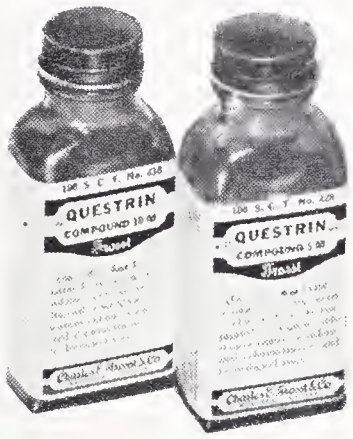
Natural HORMONE THERAPY

"QUESTRIN" PLAIN

Brand of naturally occuring, water soluble, arally active estrogenic conjugates (equine).

"QUESTRIN" COMPOUND

with vitamin B compound as a PLUS factor.



Orally active, highly potent and well-tolerated, QUESTRIN promptly relieves estrogen deficiency symptoms . . . and patients enjoy a feeling of physical and mental well-being that is not obtained from synthetic preparations.

QUESTRIN COMPOUND contains B complex factors, proven essential to the proper metabolism of steroid hormones . . . an important consideration in the treatment of menopausal symptoms.

"QUESTRIN" PLAIN
Full Strength

S.C.T. No. 428 "Frosst"
Coloured Red

Each tablet contains:
Questrin, equivalent to the activity of
Sodium oestrone sulphate..... 1.25 mg.

"QUESTRIN" PLAIN
Half Strength

S.C.T. No. 427 "Frosst"
Coloured Brick

Each tablet contains:
Questrin, equivalent to the activity of
Sodium oestrone sulphate.....0.625 mg.

DOSE

One to three tablets daily.

MODES OF ISSUE

Bottles of 20 and 100 tablets.

QUESTRIN and QUESTRIN COMPOUND afford prompt relief from all estrogen deficiency symptoms — amenorrhea, functional uterine bleeding, vaginitis, kraurosis vulvae, postpartum engorgement of the breast.

"QUESTRIN" COMPOUND
Full-Strength

S.C.T. No. 430 "Frosst"
Coloured Pink

Each tablet contains:
Questrin, equivalent to the activity
of Sodium oestrone sulphate.....1.25 mg.
plus
Brewer's yeast concentrate..... 100 mg.
Thiamine HCl..... 3 mg.
Riboflavin..... 2 mg.
Niacinamide..... 10 mg.
Pyridoxine HCl..... 1 mg.
Calcium *d*-pantothenate..... 5 mg.
Ascorbic acid..... 25 mg.
Vitamin D..... 500 I.U.

"QUESTRIN" COMPOUND
Half-Strength

S.C.T. No. 429 "Frosst"
Coloured Orange

Each tablet contains:
Questrin, equivalent to the activity of
Sodium oestrone sulphate.....0.625 mg.
plus
the same factors listed under S.C.T. No. 430.

Complete therapeutic information on request.

Frosst
SINCE 1899
THE SYMBOL OF PROGRESS
IN PHARMACEUTICAL RESEARCH

Charles E. Frosst & Co.
MONTREAL CANADA

For the Treatment of

CHRONIC ARTHRITIS

"OSTOFORTE" COMPOUND

Brand of high potency Vitamin D capsules

The treatment of chronic arthritis with massive doses of vitamin D, while entirely empirical, has proved beneficial in a great number of cases.

In many arthritics, diet may be restricted for therapeutic reasons or appetite diminished due to physical inactivity. Ostoforte Compound capsules supply massive doses of vitamin D and also adequate amounts of other vitamins, thus making up for dietary deficiencies of these factors.

"OSTOFORTE" COMPOUND

S.E.C. No. 671 "Frosst"

Ingredients	In each capsule	In average daily dose (4 capsules)
Vitamin D	50,000 I.U.	200,000 I.U.
Vitamin A	1,666 I.U.	6,664 I.U.
Thiamine HCl	0.67 mg.	2.67 mg.
Riboflavin	1.0 mg.	4.0 mg.
Niacinamide	6.67 mg.	26.67 mg.
Ascorbic acid	15.0 mg.	60.0 mg.

DOSE

Up to six capsules daily.

MODES OF ISSUE

Boxes of 50 and 100 capsules.

IMPORTANT: *Ostoforte Compound and Ostoforte should not be administered to patients with "impaired kidney function" nor to children in repeated doses.*

In those cases where the normal diet is adequate in Vitamin B factors and Vitamin A —

"OSTOFORTE"

S.E.C. No. 651 "Frosst"

Each capsule contains 50,000 I.U. Vitamin D.

Complete therapeutic information on request.

Frosst
SINCE 1899
THE SYMBOL OF PROGRESS
IN PHARMACEUTICAL RESEARCH

Charles E. Frosst & Co.

MONTREAL CANADA

'In your hands'

The future of Canada is in the hands of the medical profession. The young mothers of today rely on their doctor's advice on the care and proper nutrition of their infants to a greater degree than ever before.

Child-rearing is becoming more and more scientific as the years pass, and this is especially true in the matter of diet. More attention is being given to digestive troubles, allergies, and so on.

We, at Heinz, know a little about these problems, for medical men are invariably consulted before new varieties of Heinz Cereals, Strained Baby Foods or Junior Foods are approved for infant consumption and placed on the market.

Moreover, we welcome comment and criticism from the medical profession on the broad programme of advertising we devote annually to baby feeding. This publicity, which constantly stresses the importance of consulting a physician, must send many thousands of new mothers to doctors' offices every year.

Heinz

Makers of Baby Cereals • Strained Foods • Junior Foods

Factual information for physicians about *Ramses* Vaginal Jelly . . .

Wide clinical use has proved the dependability of RAMSES* Vaginal Jelly. There are three excellent reasons why this is so:

First.. Tests conducted by an independent accredited laboratory establish that no product exceeds its *immobilizing speed,* when the officially recognized test is employed.

Second.. RAMSES Vaginal Jelly provides effective *barrier action.* Julius Schmid was the *first* to use the synthetic gum, sodium carboxymethylcellulose, as a base for a vaginal jelly. This gum has been shown, by direct color photography, to have adherence properties which provide complete occlusion for at least 10 hours after application to the cervix.

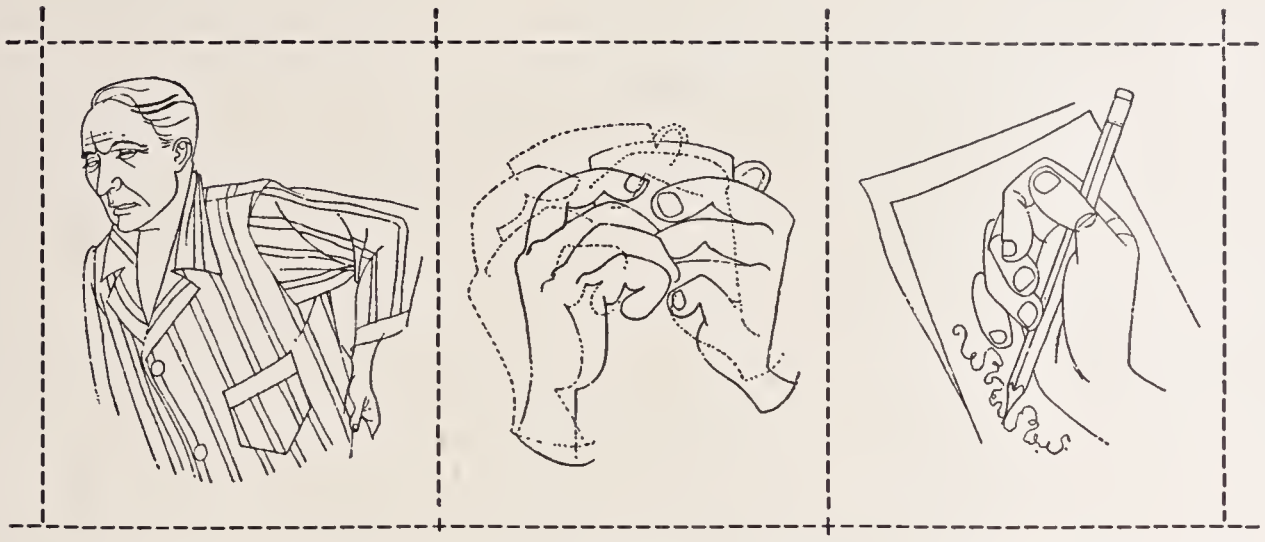
Third.. *All* RAMSES Gynecological Products, as a matter of policy, are offered for use *only* under the guidance of the physician.

**The word RAMSES is a registered trademark.*

JULIUS SCHMID (Canada) LTD.

31 Teraulay Street, TORONTO, ONTARIO

new uniform oral dosage



in muscle spasm of rheumatic disorders | in acute alcoholism | in certain neurologic disorders

The new, uniform oral dose for adults is 1-3 grams. This may be repeated 3-5 times per day.

The first dose prescribed should be at the lower end of the recommended dosage range (an occasional patient may complain of side effects when large doses are given at the start of Tolserol therapy). Subsequent doses may be adjusted to the needs of the individual patient. Whenever possible, Tolserol should be given after meals. When Tolserol is given between meals, it is desirable that the patient first drink ⅓ glass of milk or fruit juice.

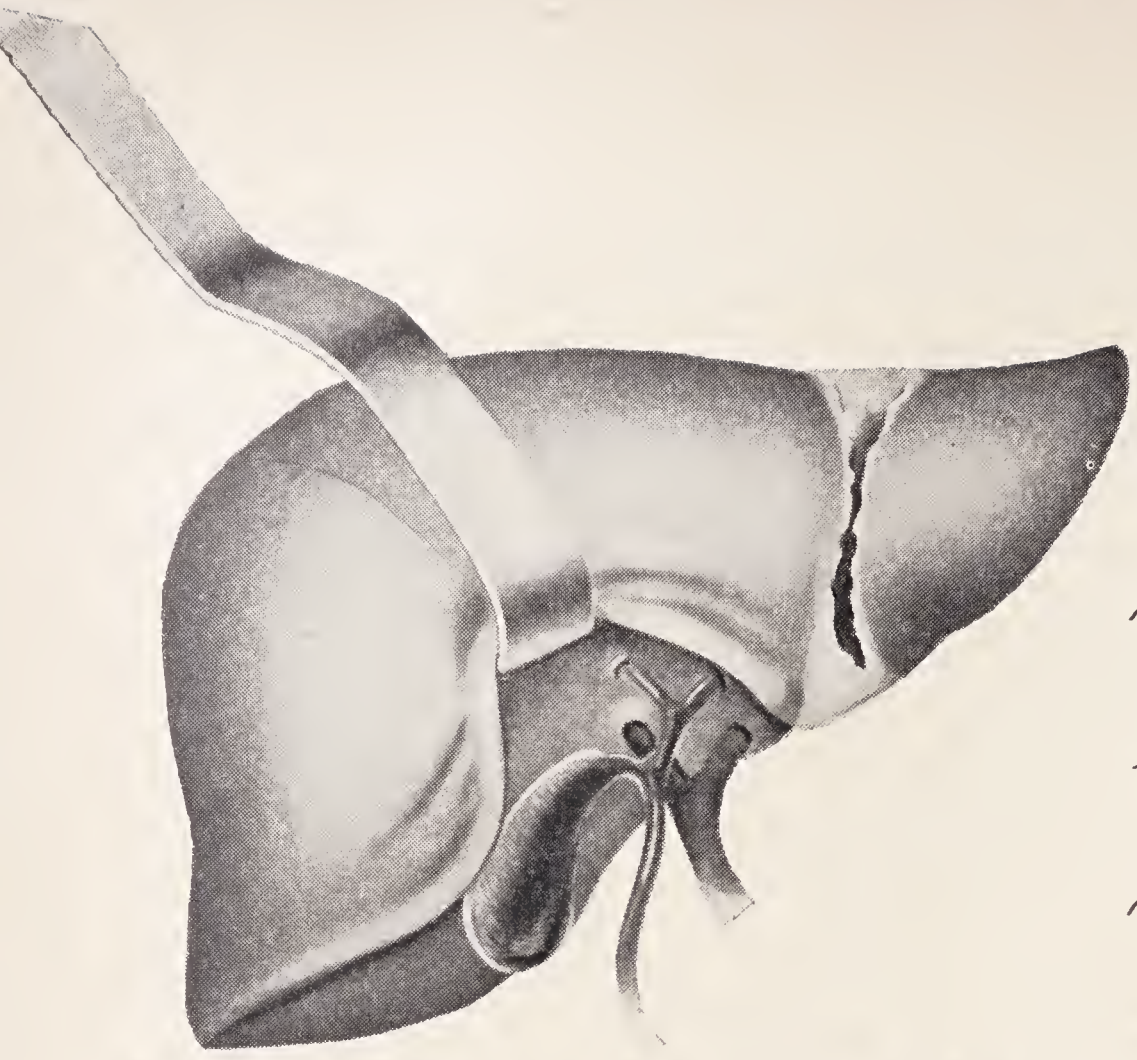
Tolserol

Squibb Mephenesin

Tablets, 0.5 Gm., bottles of 100; *Elixir*, 0.1 Gm. per cc., 16 oz. bottles; *Intravenous Solution*, 20 mg. per cc., 100 cc. ampuls.

SQUIBB

'TOLSEROL' IS A REGISTERED TRADEMARK OF E. R. SQUIBB & SONS OF CANADA LTD., 2245 VIAU ST., MONTREAL.



to restore
normal
fat
metabolism

HEPA-DESICOL®

Kapseals®

combined lipotropic and bile therapy

HEPA-DESICOL combines choline, methionine, and inositol with Desicol (desiccated whole fresh bile, Parke-Davis). It is valuable in the treatment of liver dysfunction often accompanying early cirrhosis, alcoholism, diabetes mellitus, malnutrition, obesity, and atherosclerosis.

Lipotropic action of choline, methionine, and inositol is well established; Desicol not only provides additional bile but also stimulates normal bile flow. This dual action of HEPA-DESICOL provides more effective therapy of disturbed fat metabolism.

HEPA-DESICOL Kapseals are supplied in bottles of 100 and 1000.

each Kapseal contains:

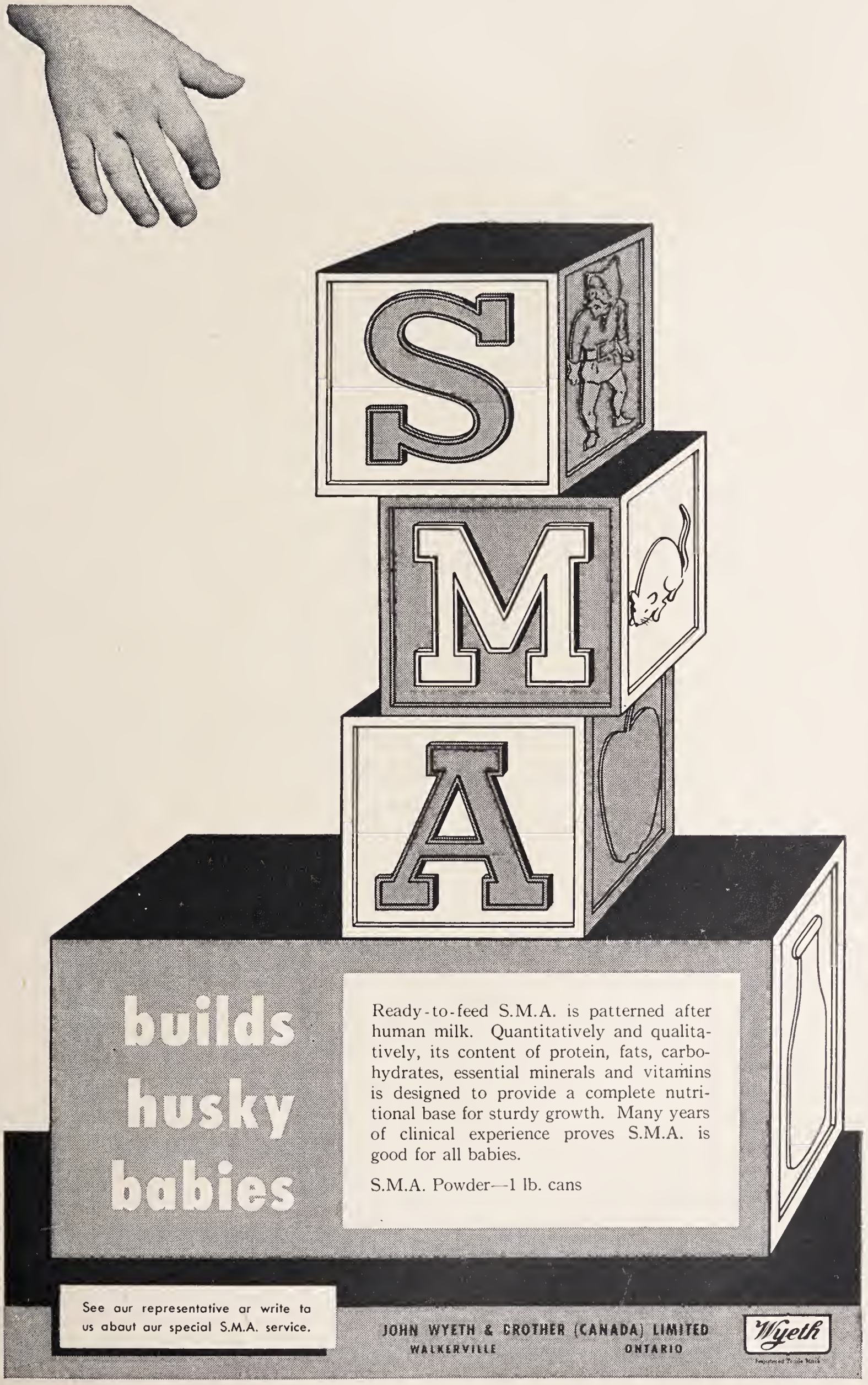
dl-Methionine	150 mg.
Choline Bitartrate	200 mg.
Inositol	50 mg.
Desicol®	150 mg.

dosage — Two to four Kapseals three times a day, with or immediately following meals.

MEDICAMENTA VERA · PD & CO.

Parke, Davis & Company, Ltd

WALKERVILLE, ONTARIO



builds husky babies

Ready-to-feed S.M.A. is patterned after human milk. Quantitatively and qualitatively, its content of protein, fats, carbohydrates, essential minerals and vitamins is designed to provide a complete nutritional base for sturdy growth. Many years of clinical experience proves S.M.A. is good for all babies.

S.M.A. Powder—1 lb. cans

See our representative or write to us about our special S.M.A. service.

JOHN WYETH & BROTHER (CANADA) LIMITED
WALKERVILLE ONTARIO

Wyeth

liver disorders

diabetes

atherosclerosis

coronary occlusion

hypertension

obesity

nephrosis

Hypercholesterolemia is often found in liver disease, diabetes, atherosclerosis and its associated coronary occlusion, hypertension, obesity and nephrosis.

Accumulating evidence shows that lipotropic therapy, as available in Methischol, will help to normalize cholesterol and fat metabolism. By reducing elevated blood cholesterol levels in most patients, lipotropic therapy may "prevent or mitigate" cholesterol deposition in the intima of blood vessels. In liver disorders, lipotropic factors reduce excess fatty deposits and encourage regeneration of new liver cells.

newly improved lipotropic formula

methischol

now contains added lipotropic vitamin B_{12}

suggested daily therapeutic dose of 9 capsules or 3 tablespoonfuls provides:

Choline Dihydrogen Citrate	2.5 Gm.*
dl-Methionine	1.0 Gm.
Inositol	0.75 Gm.
Vitamin B_{12}	9 mcg.
Liver Concentrate and Desiccated Liver	0.78 Gm.**

*present in Methischol Syrup as 1.15 Gm. choline chloride
**present in Methischol Syrup as 1.2 Gm. Liver Concentrate

Supplied in bottles of 100 and 500 capsules, and 16 oz. syrup.

Literature and samples available

Davis & Lawrence Co. (Canada) Ltd.
1910 St. Antoine Street, Montreal

U.S. Vitamin Corporation
Sole Canadian Agent of
Casimir Funk Laboratories Inc. (affiliate)